OBSTETRICS

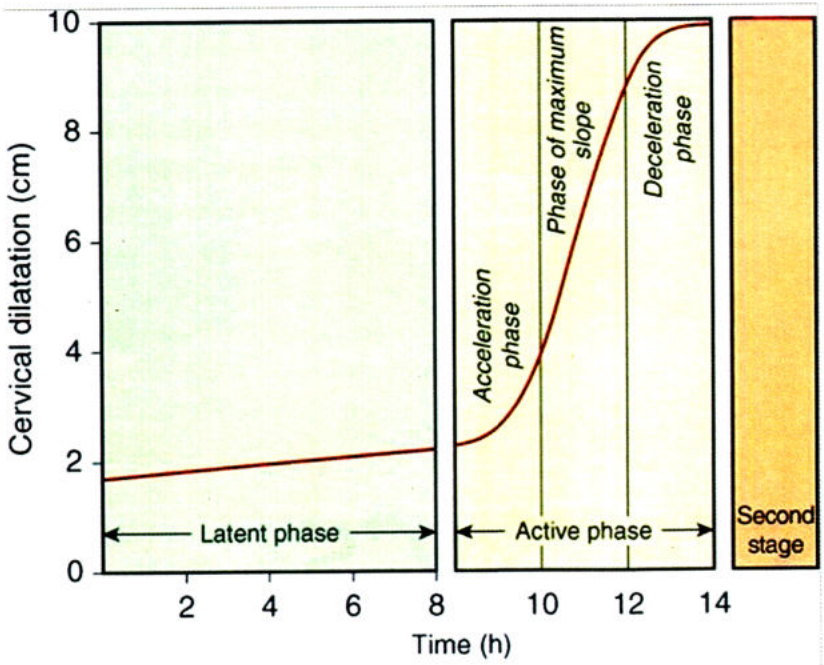
&

GYNAECOLOGY

LABOUR

Normal Labour:-

Preterm labor(<37 weeks)



First stage of labour

Latent phase
Cervix < 3 cm in dilatation

Active phase
4 cm cervical dilation

Latent phase

Irregular contractions-not very strong / painful Dilatation is not smoothly progressive

If >14 hours in Multigravidae

If >20 hours in Primigravidae

Prolonged Latent phase

Active phase:- Acceleration stage → Phase of Max→ Decelaration phase → Second stage of Labor.

→ Cervix is completely dilated in Second stage of labor.

→ 4-8cm dilatation of cervix is the strongest contraction of patient

Note:- We can stop the pre-term labor/ Tocolytic administration can only be given till latent phase of first stage of labor.

Tocolytics is used if

→ Cervical dilation <3cm

→ Gestational age <34 weeks

Causes of preterm labour

→ Infections (UTI, Asymptomatic Bacteriuria)

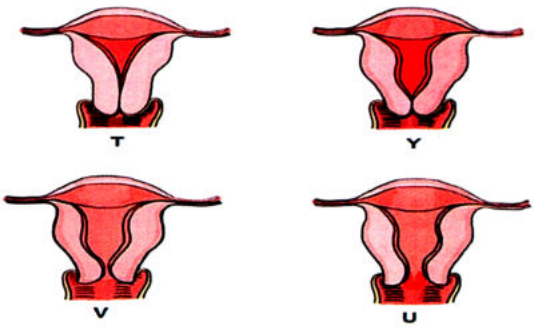
→ Chorioamniotis (PID in pregnancy)

→ Anatomical causes
- Bicornuate uterus
- Unicornuate uterus
- Cervical Incompetence

→ Overdistension of Uterus
- Twins
- polyhydramnios
- Macrosomia

Diagnosis of preterm labor on USG

At 12-13 week during the investigation of "Nuchal translucency" or "Nasal Bone scan"- we can check cervix if it is <2.5 cm– short cervix
T-shape – Normal
Y→V→U shape – progression of cervix in preterm labor.



Prediction of preterm labour by investigating vaginal secretions

→ **Fetal fibronectin**

– Amniotic sac is strucked to uterine cavity with helps of fibronectin, so fibronectin presence 22-37 weeks in vaginal secretion indicates preterm labor. Normally seen in<22 weeks and >37 weeks

Tocolytics

→ **B. Agonist**
- Salbutamol
- Terbutalin
- Ritodrine
- Isoxsuprine

– S/E –↑ ↑PR Pulmonary edema

→ **Ca-Channel blockers** – Nifedipine – DOC for preterm labour
→ **Ca-Antagonist** – $MgSo_4$ (Neuroprotective Action)
→ **PG Synthetase Inhibitors** – Indomethacin
→ **Oxytocin Antagonist** – Atosiban (No proven change in Neonatal Mortality/ Morbidity)
→ **Progesterone** – Safest Tocolytics, but used more for prophylaxis rather than treatment.
→ **Nitric Oxide donors**– Nitroglycerine patches

Note:- Tocolytics are only useful for:
- Pregnancy <34 weeks
- Cervix dilation <3 cm
- Lung Maturity (To buy time for steroids to get lung Maturity)

Lung Maturity

→ L:S Ratio >2.1 (2.3:1 accurately)
→ Best/ final/ indicator for lung maturity in Amniotic Fluid → Phosphatidyl glycerol

Note:– Most Important part of surfactant - "Phosphatidyl choline" (70% of total surfactant), that is made by Type II alveolar pneumocytes 24 weeks onwards till 34 weeks (till lungs get Matured).When lungs get Matured by 34 weeks, phosphatidyl glycerol appears that is storage form.

Assessment of Lung Maturity

→ Assess lipids in Amniotic fluid and there is formation of bubbles that suggests fat is converted into soaps by saponifying agents

→ Phosphatidyl glycerol in Amniotic fluid only (phosphatidyl choline is present in amniotic fluid, fetal serum maternal serum).

Shake test
Tap test

Assess lipids in Amniotic fluid and there is formation of bubbles that suggests fat is converted into soaps by saponifying

Nile blue sulphate test → To assessthe fat production by baby skin and assessing the lung maturity by that.

- Interpretation- Orange colour shows fat production by skin cells. If >50% cells are orange that means enough fat is produced and lungs maturity is attained.

Extra – **Nitrazine paper test** – done for PROM, as in PROM there is Alkaline Amniotic fluid in vagina

→ Interpretation
- Vaginitis → Acidic PH → Redin Colour
- PROM → Alkaline PH → blue in Colour

Ectopic Pregnancy - Incidence 1-2%

→ With ART →5%
→ After 1 Ectopic → 15%
→ After 2 Ectopics → 30%

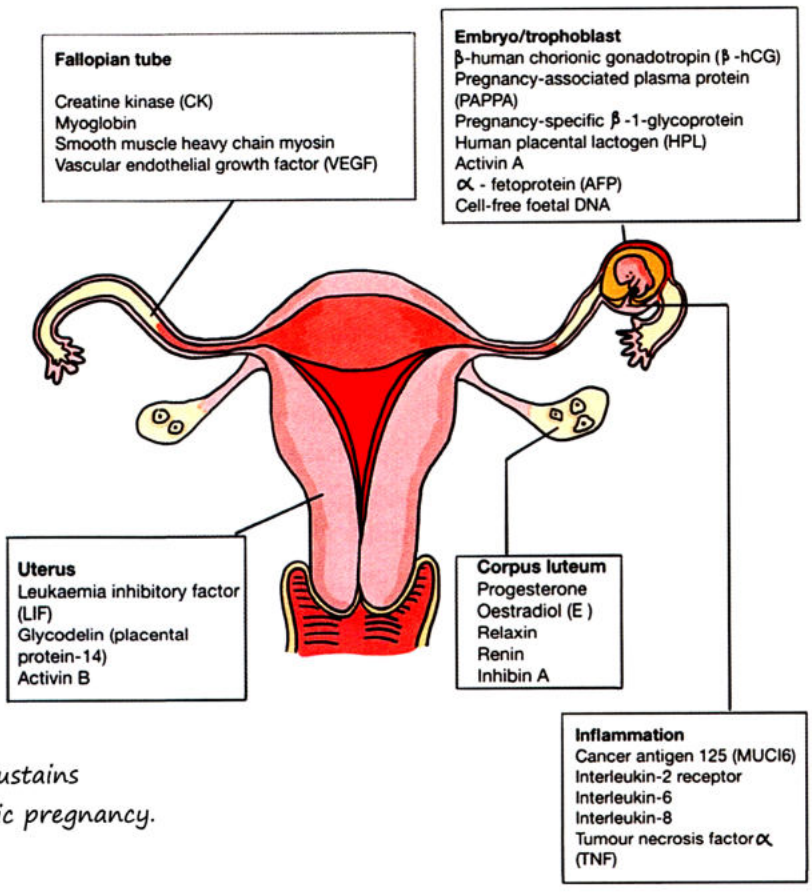
Etiology

- PID –Previous Ectopic pregnancy
- Rx of infertility
- IUCD, POP
- H/O Tubal surgeries
- H/O Endometriosis, Tuberculosis

M/C site – Tube (Ampulla – M/C)

Time of Rupture of Ectopic pregnancy

- Ampullary – 6-8 weeks (M/C)
- Isthmus → 4-6 weeks
- Cornual (Interstitial) → 12-16weeks



Note:- Progesterone of corpus luteum sustains the endometrium and the ectopic pregnancy.

Fate of Ectopic

→ M/C is vascular Insufficiency causing bleeding – due to shedding of decidual cast
→ 2nd M/C fate of ectopic– Tubal Abortion
→ Rupture of tube

Presentation

→ M/C – pain Abdomen > bleeding PV
→ Syncopal Attacks
→ Signs of shock
  - Hemoperitoneum – Cullen sign (periumblical bruising)
  - Turner sign (flanks bruising)
→ Abdominal distention

Mx of ectopic (unruptured)

→ Resuscitation + plan Sx
  - Linear salpingostomy
  - Resection and reanastomosis

Mx of Ruptured Ectopic stage

→ Laprotomy
→ If patient stable – Laproscopy
→ In cases of ruptures ectopic stage total salpingectomy is done

Medical management of Unruptured ectopic

→ Methotrexate
→ Actinomycin
→ KCL injection

Medical vs Surgical Management of unruptured ectopic.P

Medical Mx		Surgical Mx
<5000 I.U	HCG	>5000 I.U
<3.5 – 4 cm	Sac size	>3.5- 4 cm
Absent	Cardiac Activity	Present

Early diagnosis of Ectopic pregnancy

→ TVS (Trans Vaginal Sonography)-Best

	Gestational sac	Cardiac activity
TVS (-1 Week TAS)	4+ weeks (*)	5+ weeks (*)
TAS	5+ weeks (*)	6+ weeks (*)

-1 week

(* = +/- 2 to 3 days)

→ 2/3rd patients HCG is used to diagnose – Doubling time
  - Normal pregnancy – HCG doubles in 2 days
  - Ectopic pregnancy – HCG doubles at 5-7 days
→ Serum progesterone levels
  - <5ng → Missed Abortion /Ectopic pregnancy
  - >25ng → Normal pregnancy

Discriminatory zones (Values at which Sac must be seen)

- **TVS** – HCG >1500
- **TAS** – HCG >6500

Note → Pregnancies can also be present in:

→ Abdominal – Rarest, but can go to full term

→ Ovary

→ Cervix

Abdominal pregnancy →Mx is Laprotomy and if placenta is struck to posterior Abdominal wall → We give MTX or Actinomycin after delivery to degenerate placenta.

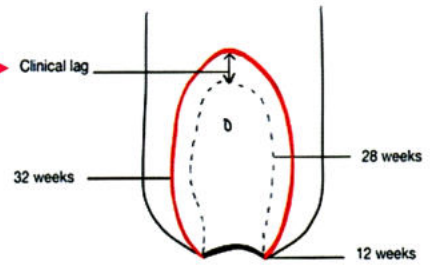
IUGR → M/C/is Idiopathic (60%)

→ Fetal causes → due to inborn errors of Metabolism/ congenital Anomalies

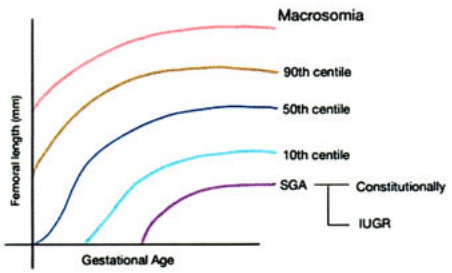
→ Maternal Causes → PIH, UTI

→ Placental causes → Fenestrated Circum vallate

Dx → Clinical lag of 2-3 weeks



→ Growth chart log



IUGR presentationEarly onset (good prognosis)Late onset (poor prognosis)

→ Chromosomal defects

→ Congenital Anomalies → Symmetrical Small babies

→ HC/AC = 1

→ Estimated fetal weight/ Femoral 3 length → Ponderal Index = 8.3, It is always maintained

→ PIH, Renal (UTI) Infections → Asymmetrical Small babies

→ HC/AC = >1

→ Ponderal Index <7

Note: Insult to fetus; first parameter to be affected → **Abdominal circumferencethenHumeral/ Femoral length** and last to be affected is **Brain.**

Management of IUGR

→ Rest in lateral position → Good Management step; because it increases venous Return ↑ - H. output ↑ ↑ → good supply to baby.

→ Adequate diet

→ Increase in Surveillance (Antepartum, Intrapartum) → Best Management of IUGR

Antepartum surveillance

→ Daily fetal movement count >10 in 12 waking Hours.

→ NST Reactive After 32 weeks – Increase should be 15 beats from baseline and lasts > 15 seconds and it should happen >2 in span of 20 minutes.

Note: - Non Reactive NST does not mean baby is having distress

Biophysical profile (BPP) by USG is the Best Method in the Antepartum Surveillance. It includes:-

→ **NST** – if Reactive -2 points.

→ **AFI** (Amniotic fluid Index) – 10-15 Normal - 2 points

Oligohydrominos –500ml or <5 AFI

Polyhydraminos–2000ml or >24 AFI

- In case we see single pocket - < 2 cm → Oligohydrominos
  > 8 cm → Polyhydrominos

→ Fetal Movement, Fetal tone, Fetal breathing

(2) (2) (2)

Total = 10 points of BPP

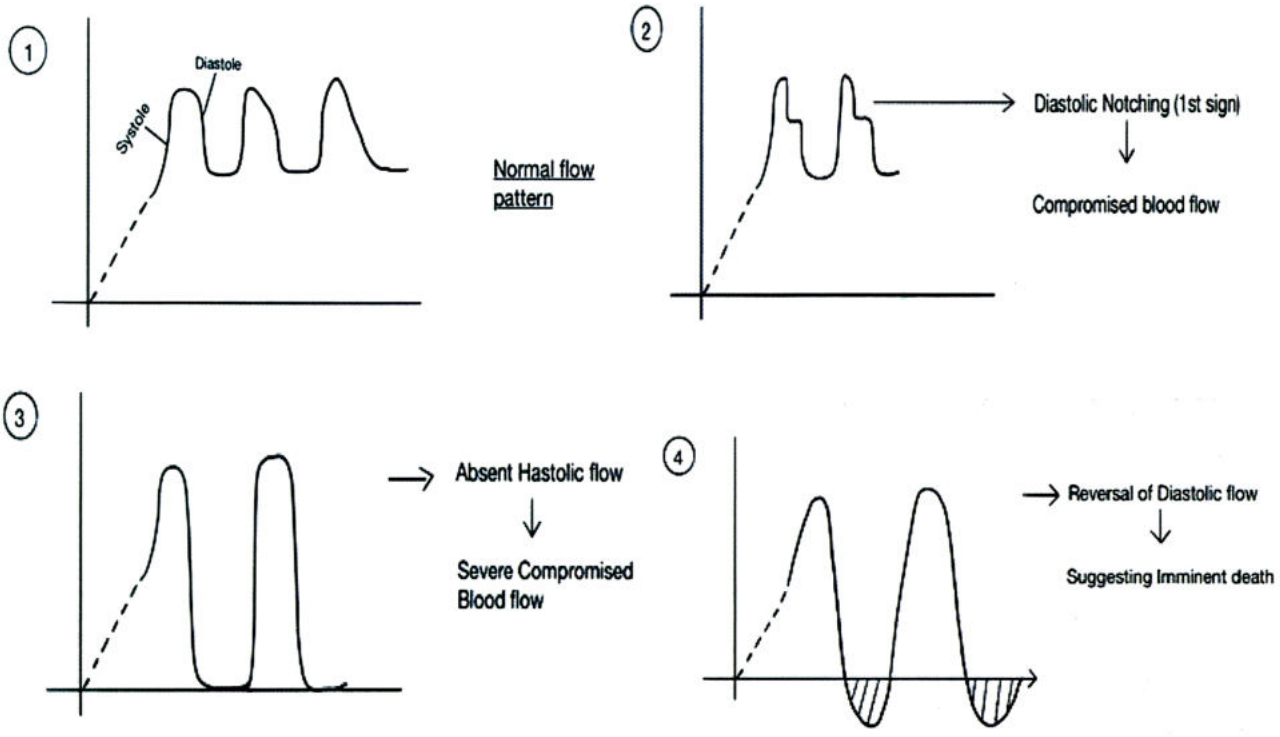
Modified BPP

→ NST + AFI

Doppler of Umblical Artery and uterine Artery

→ Flow pattern showing systolic and diastolic flow uniformly

Doppler of umbilical Artery



VAST – Vibro Accoustic Stimulation Test

→ We give vibrations with probe along with loud noise (sound box) to baby - Baby startles and wakes up form sleep – HR ↑ ↑

→ It is not a part of BPP

Contraction stress test / Oxytocin challenge test

→ Used to Assess possibility of Normal delivery in IUGR

→ We administer and watch for HR in baby with respect to uterine contractions

Interpretation: - Oxytocin administration

- Contractions causing ↓ ↓ HR of baby – Withold normal delivery
- Contractions does not cause ↓ ↓ HR of baby – Normal delivery can be done

Intrapartum – Surveillance

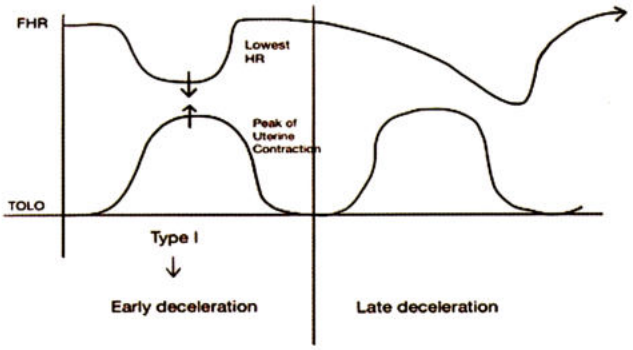
→ Fetal Heart Rate – Stethoscope, Doppler

→ Fetal Scalp pH - > 7.2 (normal)

→ Fetal ECG

→ Cardiotocography – Best

- Assessing FHR along with uterine Contractions



→ Type I – HR from baseline fall – Early deceleration is due to Head Compression n- Normal in Active Labor

→ Late deceleration – it is seen in placental Insufficiency → due to calcific Vessels

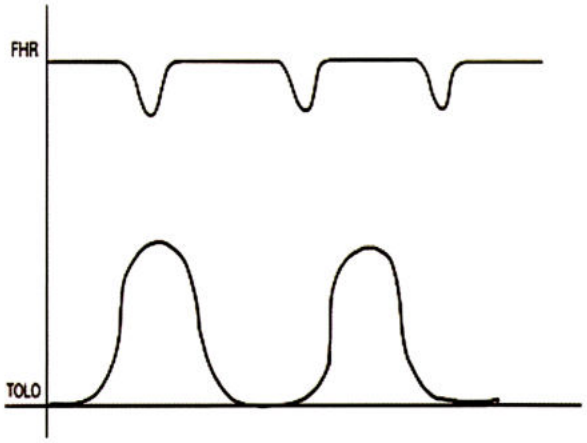
Note – Sinusoidal pattern is rare entity, but is associated with high rates of fetal morbidity and mortality

It indicates: -

- Severe fetal hypoxia
- Severe fetal Anemia
- Fetal maternal Hemorrhage

} **Immediate C-section**

→ Variable deceleration



→ Associated with umblical cord Compression

→ M/C form of deceleration and is assoc with deceleration and recovery <30 sec.

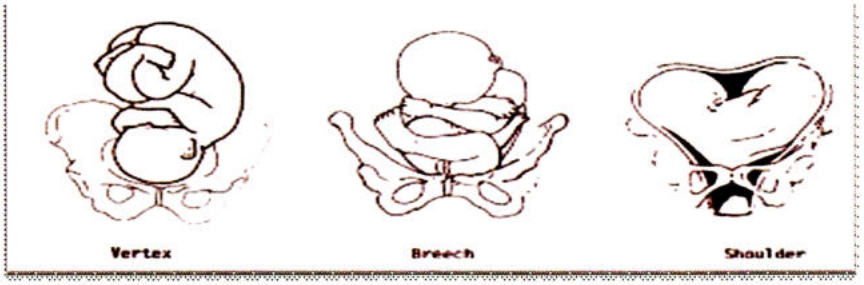
→ If it is >60sec, then it is abnormal pattern of variable decleration

Presentation– Pole of the fetus lying in the lower segment

→ Head is lower segment of uterus – Cephalic presentation

→ Breech in lower segment – Breech presentation

→ Shoulder in lower segment – Shoulder presentation



LIE – the vertical axis of baby in relation to vertical axis of mother

→ Cephalic and Breech presentation has vertical lie- Can be vaginally delivered

→ Shoulder presentation has transverse lie - Always C-Section is done, even if baby is dead.

Scenario:- At 37 weeks, uterus relaxed (woman not in labour):-

→ In Breech and shoulder presentation (transverse lie), external cephalic Version (ECV is tried, to make them cephalic.

→ Around 37 weeks liquor is reducing and baby is increasing in size so if turned, it stays in that position.

→ ECV (External cephalic version) not done around 30-32 weeks, baby can turn back to prior position.

In the case of Transverse lie

→ If patient in labour → C-section

→ If patient presents at 37 weeks (not in labour) → Try ECV

Note:- Only in 2nd Twin in T-lie (uterus is relaxed) → Internal podalic version (IPV). Otherwise (IPV) is contraindicated in T-lie due to risk of rupture of uterus.

Presenting part – the part of the presentation at internal os is presenting part.

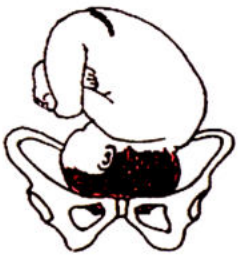
If Presentation is Cephalic

→ Attitude – Flexed Head

→ Presenting part- Vertex

→ Diameter Of Engagement – Suboccipitobregmatic 9.5cm

→ M/C positions of vertex – left occipital anterior (LOA) and left- occipito transverse (LOT).



Steps Of Labor In Normal Vertex And Flexed Head

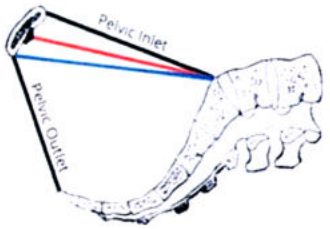
1. **Engagement** – Read enters the pelvic floor
2. **Descent**
3. **Flexion** (ongoing)
4. **Internal rotation** – head hits the pelvic floor (levator ani) and rotates internally and presenting part comes near vagina.
5. **Delivery by extension**
6. **Restitution** – Correction of internal rotation of head and neck after delivery

→ Diameter of engagement – Submentobregmatic (9.5cm)
- Submentovertical (10cm)
→ M/C position of face – Left Mento-anterior
→ In right Mentoposterior - diameter of engagement is Sternobregmatic (17.5cm) – cannot deliver.
→ Mentoanterior –face delivery happens
→ Mentoposterior
- If it becomes mentoanterior – Delivers
- If stays mentoposterior – C-Section

Pelvic Inlet

Pelvic diameters

Palpate the sacral promontory with tip of middle finger and mark the level of lower border of pubic symphysis at base of thumb. This distance between tip of middle finger to base of thumb is diagonal conjugate (DC).



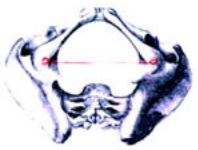
Diagonal conjugate → distance between lower part of symphysis pubis and top of the sacrum → 12cm
Anatomical conjugate → distance between upper part of symphysis pubis and top of sacrum → (DC – 1cm) = 11cm
Obstetrical conjugate (OC) → From pubic tubercle to top of sacrum→ (DC -2cm) = 10cm
- Narrowest diameter of pelvic inlet
- If O.C<10cm – Contracted inlet

Mid Pelvis

From lower border of symphysis pubis to ischial spine laterally and S_4- S_5 posteriorly is plane of mid pelvis.
→ (ISD) Interspinous diameter is the narrowest (10.5cm) and is station O
→ When Biparietal diameter (BPD -9.5cm) negotiates the ISD, that time occiput felt per-vaginally is at +2 station (2cm below ischial spine)
→ In Adequate pelvic – baby easily reaches +2 station and beyond
→ ISD <10 cm -Small pelvis, <8cm - Contracted pelvis

Outlet

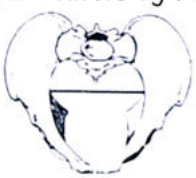
Distance between ischial tuberosities <8cm – contracted outlet (outlet contracture)



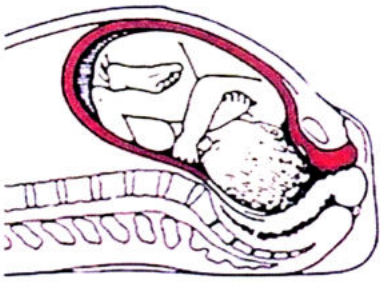
Types Of Pelvis

Gynecoid pelvis
- M/C type
- Rounded cavity
- OA Presentation is seen

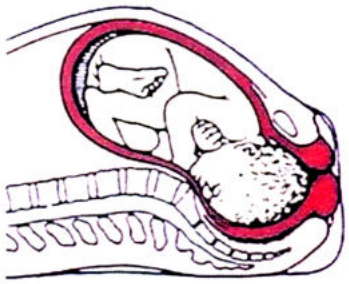
Anthropoid pelvis
- Slightly narrow side walls
- OP in anthropoid has no space to rotate
- So, it delivers by Face to Pubis



7. **External rotation** – rotation of head outside due to shoulder rotation inside (women posterior hits the pelvic floor.



Engagement; descent. flexion,



Further descent, internal rotation

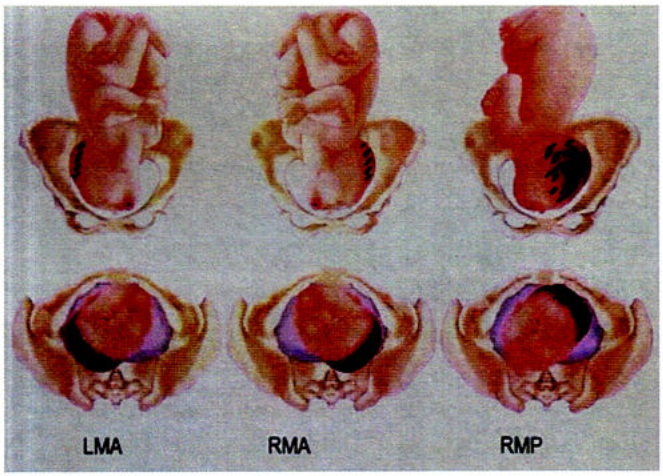
Complete extension.

Restitution & external rotation

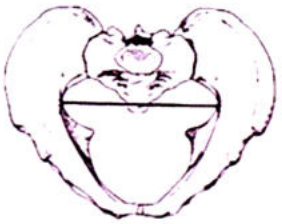
→ MC malposition is right occipitoposterior – (ROP)
→ MC presentation is cephalic
→ MC malpresentation is breech
→ 80% times OP (occipitoposterior) → becomes OA → Delivers normally
→ 15-16% times OP → Stay OP → delivers as Face to Pubis.
→ 2-3% becomes –Occipitotransverse (OT) and gets stuck and known as Deep Transverse. Arrest (DTA)
→ In DTA – Can try manual rotation and extract by forceps and best managed by "C-section".

Deflexed Head -(Straight head)
→ Presentation –Brow
→ Diameter of engagement – Mentovertical (MV) (14cm)
and always do C-Section.

Extended Head - Presentation -Face



Android pelvis



- Male type of pelvis
- Spacious posteriorly, becomes narrow anteriorly
- OP in android can rotate to OT, but cannot rotate to OA.

So, DTA is seen

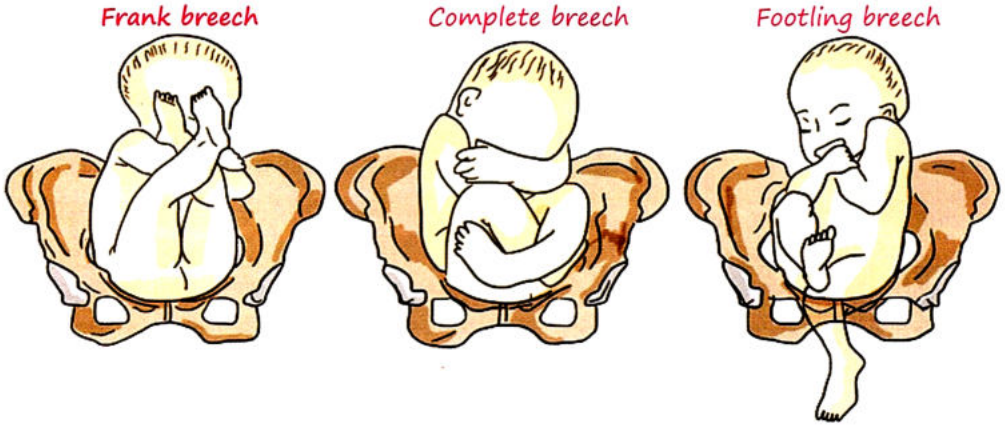
Platypelloid/Flat pelvic –

- Rarest type <3%
- Oblong compressed from front and back
- Face presentation is seen

Breech

→ 3% at term

→ Types



- Flexed (Complete) Extended (Frank) - Can deliver normally.
- Footling (Incomplete) → C-Section

→ MC type of breech – Extended breech

→ Best type of breech for vaginal delivery – Extended Breech

→ Type of Breechvaginal delivery done – Assisted Breech Vaginal Delivery:- Here we do not touch the baby till umbilicus is seen and delivers on own.

→ For Extended legs in Breech delivery – Pinard's Maneuver can be done – Middle/index finger is used to tap the popliteal fossa at legs and flexing of extended legs is done.

→ For Extended arms – Lovset's maneuver

→ For After coming head

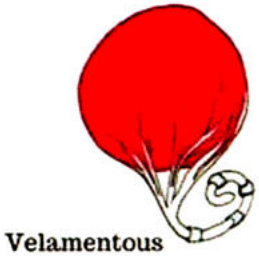
- Can do Malar flexion and shoulder traction
- Jaw flexion and shoulder traction known as Mariceau Smellie Viet (MSV)
- Pipers forceps can be used → Safest
- Burns Marshall Technique

Note:- **Burns Marshall Technique** – Baby allowed to hang by its own weight and when nape of neck is visible, both feet are held and swing in a long arc upwards. Head is born by Flexion in this maneuver.

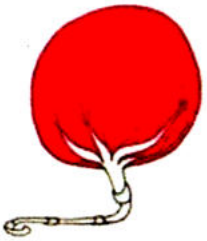
Variations of placenta

Normal placenta – Two Arteries and one vein – Normallyright vein gets obliterated.

Types of placenta –

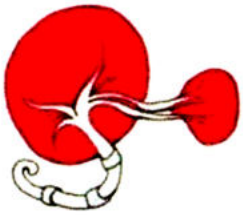


Velamentous Insertion

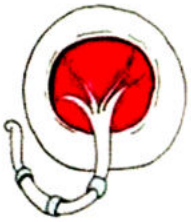


Marginal Insertion

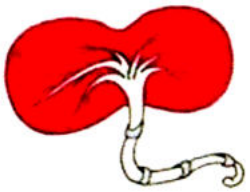
Circummarginate



Succenturiate Lobe



Circumvallate



Bilobate

→ **Marginal placenta** aka Battledore placenta

→ **Succenturiate placenta** – can present with Secondary PPH

→ **Bilobed placenta** → associated with polyhydraminos and can also have Abruption

→ **Circumvallete Placenta** → Extra chorial placenta – Doubling of Amnion and chorion around periphery of placenta

→ **Circum marginate Placenta** → Also type of Extra chorial placenta.

- Thin membrane along with fibrin deposits around the periphery.
- Association with Abruption and IUGR.

→ **VelamentousPlacenta** → There is problem of Torrential Hemorrhage of fetal origin while delivery in Vasa previa

- Velamentous Cord when present at OS. It is called "Vasa Previa"
- In >50% cases of vasa previa, baby dies.

Note: - Doppler in Third Trimester can pick up vasa previa best

Scenario: - Distinguish between Maternal blood and fetal blood in case of bleeding PV→to rule out either it is placenta previa or Vasa Previa → **APT test** is done (Qualitative test)

Blood sample → Alkaline denaturation of Hb
NaOH

Fetal RBC Resists Alkaline denaturation of HB

↓

Colourless/Yellow blood sample

↓

Maternal blood loss confirmed

→ Quantitative test for Assessing fetal blood → **Kleihauer Betke test** - Done for fetomaternal Hemorrhage Assessment in Rh Iso Immunization.
Ex: - Mother (A-) and baby (A+) → Mother get contaminated with "D" (Rh) antigen from blood of baby and Anti-D Ab's are made, then in next pregnancy
all Anti D-Ab are going to act against baby with D (Rh) antigen present resulting in Fetal RBC Hemolysis → Hydrops fetalis.

Mx - Anti-D administration after delivery of first baby – within 72 hours
Dose = 300mg Anti-D will neutralize 30ml of fetal blood/15ml off fetal RBC

KB test

→ We get fraction of fetal origin cells in slide with respect to Maternal RBC → We can adjust the dose of "Anti-D" according to that.
Anti-D is also given prophylactically at 28 weeks – 1st dose

↓

At 34 weeks – Another dose

↓

After delivery – another dose within 72 hrs.

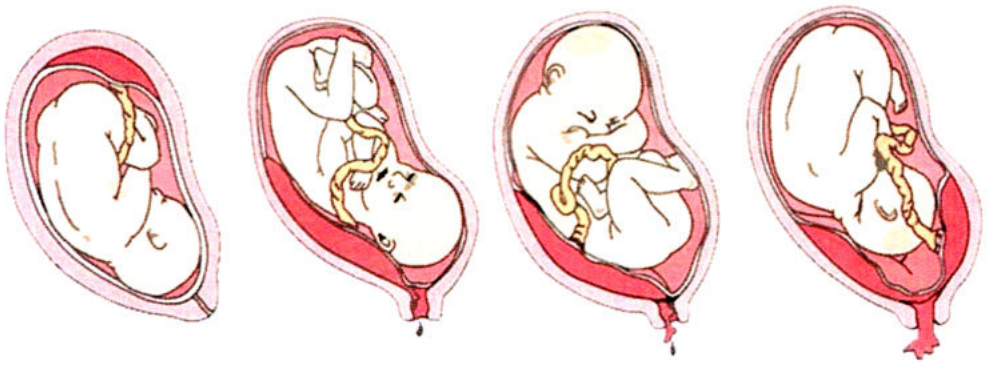
According to American guidelines: 1st dose given at 28 weeks → 2nd dose at 72 hrs after delivery.

Ante Partum Hemmorhage - any bleeding in genital tract after 28 weeks till delivery.

Causes

→ Vasa previa
→ Placenta previa
→ Abruption etc.

Placenta previa



→ Complete and partial cowering OS are major types and causes bleed
→ If placenta is in lower segment but ≥ 2 cm away from OS – Low Lying Placenta.

Scenerio:-

→ Term pregnancy (37-42 weeks) Complete/ Partial PP + bleeding → Mx = Cesarean section
→ Term pregnancy + PP (no bleed) → If Totally Covering OS → Mx = Cesarean section
→ if Partially Covering OS → PV Examination done under Anesthesia (Under TIVA/light GA) in OT

- If placenta is not present within 10cm range of PV examination that is placenta is moved away due to effacement → Mx = Trial of Normal Delivery

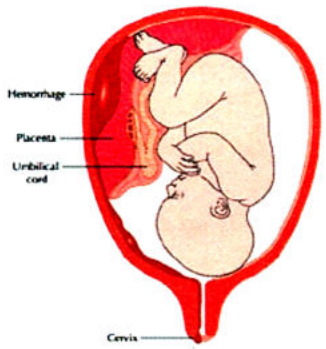
→ PP + bleeding at ≤34 weeks of gestation → Mx Rest, sedation and observation in high Risk ward after resuscitation of patient - "Mcaffee Jhonson Regime"

Note: -
- 90% of bleeding stops after first episode of PP bleeding but if bleeding continues → C. Section is done.
- Steroids are given ASAP when patient come for lung maturity
- Tocolytics are not given in bleeding PP patient and also not given in vasa previa.

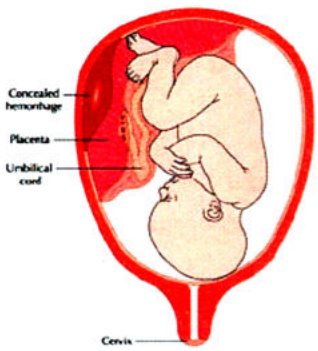
Abruption placenta

→ Separation of placenta abruptly before the delivery of baby.
→ Uterus is tender to touch

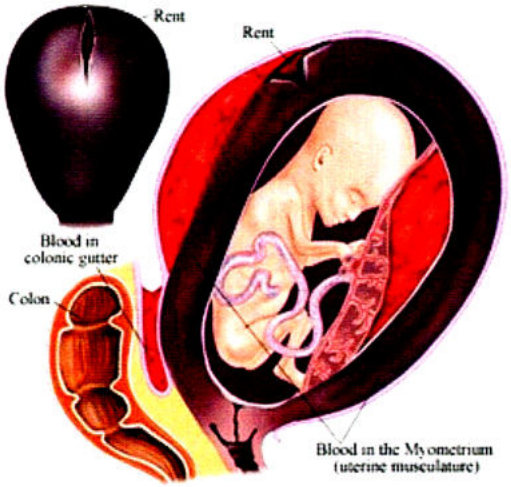
Revealed Concealed



Covulaire/Bruised Uterus



→ Uterus is tender to touch



Mx =

→ Abruption at Term → Never CS
→ If Abruption along with fetal distress (HR<110) (Scalp pH <7.2)→ CS is done

ARM (Artificial Rupture of Membranes) ↓ bleeding because of compression of the placenta. It also releases local prostaglandins that helps in Induction of Labour. Oxytocin is also administered along with it.

→ If Abruption at ≤34 weeks and blood collecting at back of placenta will release tissue Thromboplastin → Triggers coagulation cascade → DIC occurs.

Mx = Steroids → For lung Maturity + ARM is done for Augmentation of Labour.
Tocolysis is C/I in it (we hasten the delivery in Abruptio placenta)

Note:- If any Maternal problem regarding well being of mother or fetal distress – CS is done

Abruptio placenta
Placenta previa
Vasa previa

} Tocolytics are C/I in APH.

HTN in pregnancy

BP>140/90 on >2 Occasions after 20 weeks of gestation
↓ + Protenuria (>300mg of protein in 24 hour urine or 1+ or dipstick)
Pre-Eclamptic Toxemia
↓ + Convulsion
Eclampsia

Acute on chronic HTN:-

→ chronic HTN + after >20 weeks of pregnancy → superadded HTN of pregnancy
→ Low platelets < 1 lakh
→ New onset proteinuria

} happens only in pregnancy, not present before

Mx of HTN

→ DOC is Labetalol – Chronic HTN and Hypertension of pregnancy (100-200mg – Thrice a day).
Labetalol IV is DOC for HTN emergency also
→ Methyldopa - 250-500mg QiD (S/E - depression and drowsiness)
→ DOC for Eclampsia – $MgSO_4$
- Pritchard regime – IV + IM
- 4gm 20% IV slowly in 3-4 minutes
- 5gm (5%) in each buttock

Note:- Most important step in Eclampsia – Delivery of baby. CS is NOT C/I
$MgSO_4$ is administered for 24 Hrs after delivery or 24 hours of last convulsion whichever is later.

5g over 4 hours is given	→ Knee jerk should be present
keep check	→ RR >14/min
on these signs	→ Urine output >100ml/4hours

- if these signs are absent then discontinue $MgSO_4$

Etiology of Eclampsia – Vasospasm of placental vasculature causes ↓ in Intra Vascular volume → Mother BP increases to give more perfusion to fetus – to overcome placental vasculature vasospasm

High BP causes:-

→ Retinal Hemorrhages
→ Subcapsular Hematomas of Liver → Epigastric pain
→ HTN Nephropathy → Proteinuria
→ Extrudes electrolytes from membranes in brain → Irritates Meninges causing Convulsions.

Vasospasm in Placental Vasculature is due to non development of trophoblastic layer in smooth muscle layer in tunica media of Blood vessels of placenta that generally occurs at 20 weeks of gestation.

Note: - Furosemide is not given/ C/I in HTN of pregnancy.

Gestational Diabetes Mellitus

→ Around 24 weeks or beyond - ↑↑ sugars (glucose)

→ Occurs due to HPL that has Insulinase like Action and ↑↑ Insulin resistance in pregnancy causing high sugars.

→ Pre-existing ↑↑ Sugars before pregnancy → Overt diabetes

→ 1st Trimester High Sugars directly proportional to Anomalies

Note: -

→ GDM does not cause anomalies it is the overt Diabetes that cause Anomalies

→ Metabollic problems, Shoulder dystocia, large babies these are common to both – GDM and overt diabetes.

Screening for Overt diabetes

→ FBS

→ HbA_1C <6.5 (Normal)

Screening for GDM:- "**1 Step Test**"by American Diabetic Association –

Fasting blood glucose (<92 is normal)

75 gm glucose given

- Normal value < 180 in 1 Hr
- Normal value <153 in 2 Hr

Any abnormal value with respect to normal values – confirms GDM

Effect of Diabetes on Mother: -

→ ↑↑ obstetric Injuries

→ ↑↑ CS/↑ Forceps and Vacuum deliveries

→ Shoulder Dystocia

→ Association with PIH in 25% cases

→ PROM, Preterm Labour

→ Chorioamnionitis

→ Puerperal sepsis – Abortion

Problems of Newborn due to Diabetes:-

→ Hypoglycemia

→ Hypocalcemia

→ Hypomagnesemia

→ Polycythemia

Anomalies in Prexisting diabetes/ overt Diabetes: -

→ M/C group of Anomalies – Cardiac group of Anomalies (TGA – Most specific VSD, PDA)

→ NTD- most specific – "Caudal Regression Syndromes" aka Sacral Agenesis , and Anencephaly

Mx of Diabetes

→ DOC – Insulin

→ OHA (Glyburide, Metformin)-Approved for use in GDM.

Extra:-

→ M/C Heart disease in Pregnancy - RHD

→ M/C lesion – Mitral stenosis- Mx :- Around 2nd Trimester Ballon Valvotomy

→ Failure of Heart disease in Antenatal Time is M/C in 30-32 weeks (CO↑ by 50%) and in Post Natal time -1st 24 Hours (Co↑ by 70%)

Note:-

→ Frusemide after delivery ↓ preload and decreases chances of Heart failure.

→ DOC for PPH = Oxytocin and avoid giving Methyl Ergometrine because it causes immediate. Contraction of uterus → ↑↑ BP → Heart failure

→ In Labour avoid straining in 2nd stage. Use forceps /vaccum → to cut short the second stage of labour/valsalva maneuvre.

→ Normal delivery is allowed in all Heart diseases and Morphine is known to decrease incidence of LVF.

→ Epidural Analgesia and Morphine Administration in Heart failure patients is beneficial.

Heart Diseases C/I for pregnancy

→ Eisenmengher

→ Primary pulmonary HTN

→ Marfan Disease - Involving Aortic Root

→ Severe Aortic Stenosis

Note: - Any Heart disease which comes in NYHA III and IV is C/I for pregnancy

- Co-arctation is not a C/I of pregnancy can be delivered by C-section.

Epilepsy in pregnancy:- does not cause Fetal Anomalies

Fate of epilepsy patients in pregnancy:-

→ 30% cases- more convulsions

→ 20% cases- reduction in convulsion

→ 50% cases- Unchanged.

In case of epilepsy in pregnancy:- Lamotrigine (DOC), Levetiracetam

Malaria in pregnancy

→ Worse Prognosis

→ ↑ Chance of fulminant hepatic failure

Mx:- DOC – Chloroquine, Artesunate & quinine in resistant cases.

Appendicitis in Pregnancy

→ ↑ rupture/ perforation/ preterm labor/sepsis- so early decision to undergo Sx needed.

Rheumatoid Arthritis in Pregnancy - Better Prognosis

Sarcoidosis → better in pregnancy

Ulcerative colitis → Unchanged in pregnancy

TB in pregnancy

→ 1st/2nd /3rd /Puerpurium - Worst prognosis ,In puerperium – worst as there is immuno suppression. Also ↑ demand (lactation) ↓ supply; heat, humidity.

→ More common in low Socio Economic Status

Abortions

Spontaneous .A → Anything delivering before <28 weeks or less than 500 gms.

Note

→ Any baby delivery > 28weeks – viable baby

→ Term pregnancy → 37 -42 weeks

→ MTP → Medical termination of pregnancy < 20 weeks

1st trimester Abortion (12 weeks)

→ Mostly because of chromosomal causes

- Trisomy 16, 13, 21 (16 M/C Association)
- Monosomy 45 x O

2nd Trimester Abortion – mostly due to Anatomical causes:-

- Incompetent OS
- Septate uterus
- Bicornuate uterus
- Unicornuate uterus

Note → Causes leading to Abortion in any Trimester:-

- Diabetes
- Hypothyroidism
- SLE
- Torch
- Syphilis
- APLA syndrome

Recurrent pregnancy losses: -

→ 3 losses at any time of pregnancy-M/C/C → Chromosomal

→ Other causes

- Anatomical
- DM/ APLA/ SLE / Hypothyrodism

It can never be due to TORCH group infection; because once they cause pregnancy loss then the women becomes Immune to the infection.

APLA – Antiphospholipid Ab syndrome

Ab against phospholipid membranes → causing thrombosis and blood vessels will get plugged / Thrombosed causing death of baby.

Thrombophilias

→ **Inherited**

- Anti-Thrombin deficiency
- Factor V Leiden mutation
- Protein S and C deficiency
- Prothrombin gene mutation

→ **Acquired**

- APLA
- Major Sx. / Immobilization
- Malignancy

Diagnosis of APLA

Clinical criteria	→ Vascular thrombosis: 1 arterial, venous, or small vessel thrombosis. → Pregnancy morbidity ≥ 1 fetal death (at or beyond the 10th week of gestation) ≥ 1 premature birth before the 34th week of gestation because of eclampsia, severe preeclampsia, or placental insufficiency ≥ 3 consecutive (pre) embryonic losses (before the 10th week of gestation)
Laboratory criteria	→ Lupus anticoagulant positivity on 2 occasions at least 12 weeks apart. → Anticardiolipin antibody (IgG and/or IgM) in medium or high titer (i.e., >40, or above the 99th percentile), on two or more occasions at least 12 weeks apart. → Anti-2-glycoprotein-I antibody (IgG and/or IgM) in medium or high titer (i.e., above the 99th percentile) on two or more occasions at least 12 weeks apart.

Note:- Definite APS is present If at least one of the clinical criteria and one of the laboratory criteria are met.

Anatomical defects

→ Only indication of unification of a Bicornuate uterus

Abortion → Recurrent Abortions

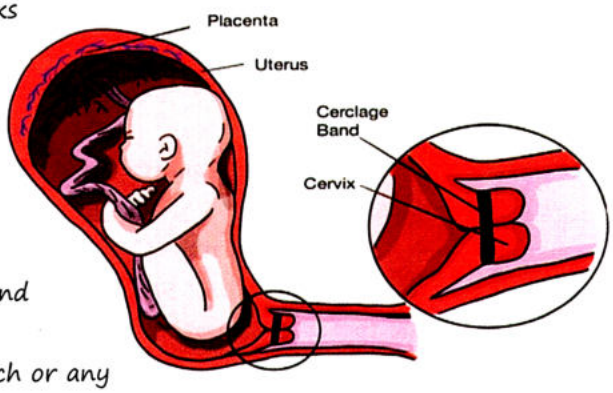
→ Septal resection is also done in bicornuate uterus

→ In Cervical incompetence, abortion occur at 20-24 weeks and there is painless dilatation of cervix.

- It can be diagnosed Antenatally by passing Hegar's Hlator (8 French).
- It is also diagnosed with Antenatal Scan at 10-13 weeks - Cervix length <2.5 cm (short) leading to Abortion or Pre-term Labour

Mx of cervical incompetence – Cerclage by 12 weeks and beyond, M/C used method is Mcdonalds method.

Cerclage Correction of the cervix



→ Shorter cervix or Mutilated cervix – Mx Shirodkar's stitch or any Abdominal cerclage -removal of cerclage is done at > 37 weeks, or when patient comes with labor.

Presentation of Abortion

→ Bleeding (most commonly),Pain

→ Bulging of Membranes

- On upper vaginal examination if OS is closed – **Threatened Abortion**
- On PV if OS is opened and products are bulging – **Inevitable Abortion**
- If OS opened, H/O passage of products and still few products of pregnancy felt through PV → **Incomplete Abortion**
- If closed, uterus is of N size and H/O passage of products → **Complete Abortion**
- If there are no symptoms of Miscarriage, and dead fetus/ Embryo is retained in uterus (baby was alive before now dead) → **Missed Abortion**

Blighted Ovum

→ On USG only gestational sac is seen, and on repeated USG After few weeks gestational Sac increases in size but no yolk sac formation fetus formation. There are missed events yolk sac formation → Fetalnode formation → Cardiac Activity

→ aka Anembryonic Gestation

MTP- Act passed in 1971 and implemented in 1972.

→ Can only be done in govt. Approved Centre

→ Done by

→ Gynecologist

→ Doctor - Trained for 6months

- Done 25 Abortions under Supervision of gynecologist.

→ 1st Trimester Abortions upto 9 weeks, we can do it by medical Abortion by

- Methotrexate
- Mifepristone followed by Misoprostol (800g) (200mg). Success rate of Mifepristone/Misoprostol:-
  - First 7 weeks – 99%
  - First 9 weeks – 95%

→ Suction / Evacuation is done ideally → 8-10 weeks

→ Dilation/ Curretage is done ideally → 8- 12 weeks

→ If pregnancy >12 weeks – DOC is Prostaglandins

- Misoprostol – PGE_1 – Tablet and used as vaginal, oral Recta, sublinguial
- Dinoprostone – PGE_2 – Gel from and given in vagina
- Carboprost – $PGF_2\alpha$ - Injection from – given im.

→ Laminaria Tents→ Act by hygroscopic method

→ If there is Abortion failure after Prostaglandins administration and to remove the dead/ Macerated fetus – Hysterotomy is done.

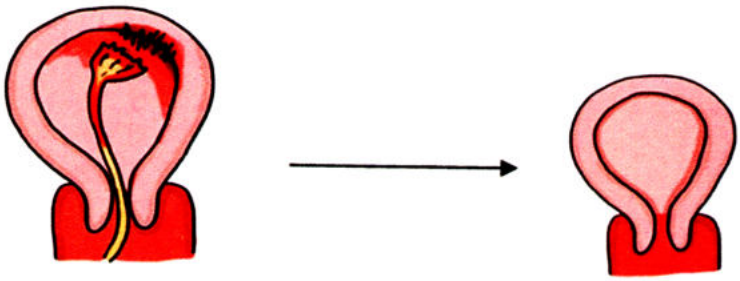
Stages of Labor

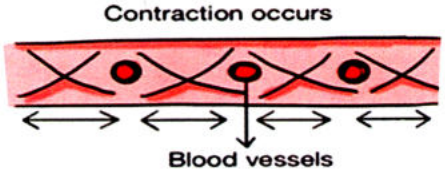
I - Contraction

II - Full dilatation of cervix → delivery

III - Delivery of placenta

IV - 1 hour Observation – to prevent PPH





Post Partum Hemmorhage

→ Any bleed genital Tract after delivery
→ M/C/C → Tone deficiency (Atonic uterus), mostly due to Trauma (Cervical, uterine, vaginal)
→ Other causes
- Thrombin deficiency (Coagulation defect)
- Retained Tissue (placental bits)

→ Any bleed > 500 ml in Normal delivery or >1000 ml in CS is PPH.
- If > 2000ml blood loss – Severe PPH
- 1000 – 2000 is moderate PPH

Note →Any bleed which reduces Hb by 1gm % is PPH.
→M/C /C of Maternal Mortality – Obstetric Hemorrhage (M/C – PPH)

Management of PPH

Prevention:-

→ Oxytocin 5-10 IU / IM (oxytocin is stored in Refrigerator)
→ Control Cord Traction
→ Massage of uterus

Mx of PPH:-

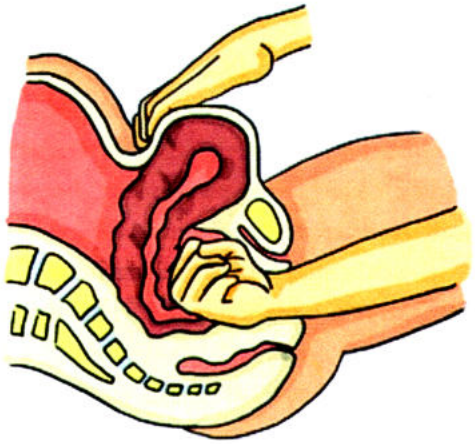
→ DOC – Oxytocin 10-20 IU by IV drip
→ Methylergometrine – IV/IM 0.2, it is C/I in Pre Eclampsia toxemia, HTN, Twins (before delivery of 2nd baby) Rh- ve pregnancy, Heart disease (MR, MS, VSD)
→ Carboprost – $PGF_2\alpha$ - 250g im only, upto 8 injections in 24 hours.
→ Misoprostol – Rectal 600-800µg
→ Activated Factor VII
→ Prophlactically - Uterine Artery Embolisatin and Intra Aortic ballooning.

→ BRACE sutures
- B. Lynch
- Hayman

→ Uterine Artery ligation
→ Internal iliac Artery ligation – Anterior division
sub ligature is done → sluggish blood flow, so promotes coagulation
→ Obstetric Hysterectomy

STOP BLEEDING

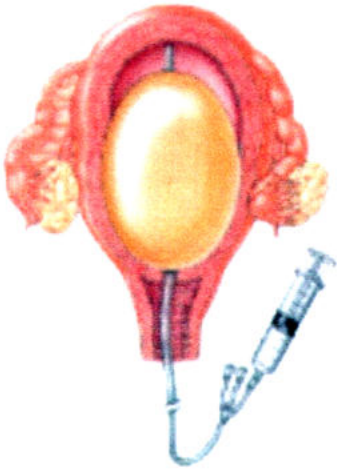
Uterine massage

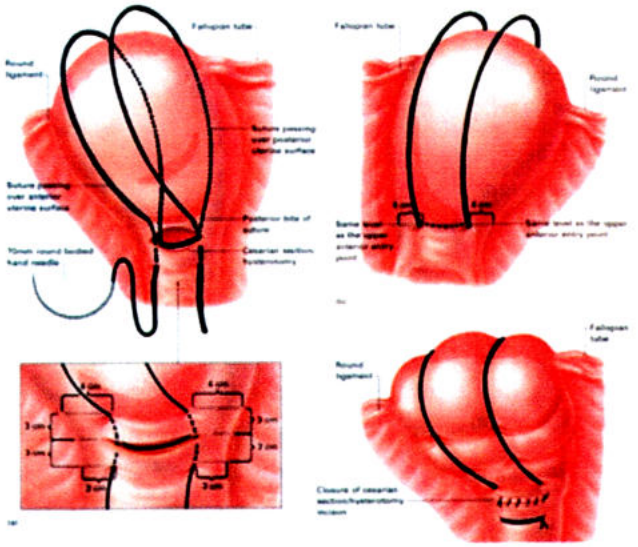


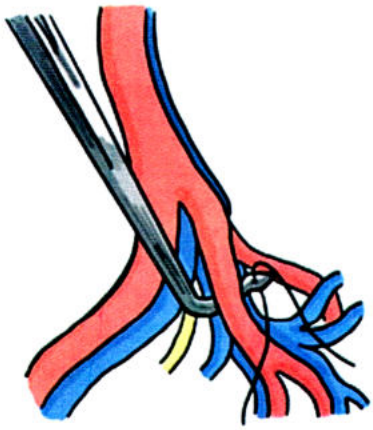
1. Syntocinon
5 units by slow intravenous injection (may have repeat dose).
(40 units in 500ml Hartmann's solution at 125ml/hour).

2. Ergometrine
0.5mg by slow I.V or I.M

3. Misoprostol
Rectal 600-1000ug.







4. Carboprost
(If available)
0.25mg by intramuscular injection repeated at intervals of not less than 15 minutes to a maximum of 8 doses (contraindicated in women with asthma).

5. Fresh Frozen plasma
4 units for every 6 units of red cells or if relentless bleeding or PT/ APTT >1.5 x normal (12-15ml/kg or total 1/L).

Exclude trauma to vulva, vagina, cervix, and uterus.

Resort to hysterectomy sooner rather than later.

Note – Dinoprostone is not given/ regular drug used to control PPH (so if all other drugs given along with dinoprostone in management of PPH except – the mark dinoprostone)

Types of PPH

1 PPH → PPH within 24 hours of delivery

2 PPH → PPH after 24 hour of delivery upto 12 weeks of delivery. It is mostly due to retained placental bits.

Mx –

→ Retained placenta → Manual Removal of placenta

→ Retained placenta bits → Curretage, S/E of curettage is Asherman's syndrome.

Removal of placenta- Best method → Controlled Cord Traction/Brandt Andrew Method.

→ Forcible separation by squeezing fundus and pulling placenta → Crede's Method- it causes lot of retained bits of placenta and that are managed by Curretage; if overly done it results in Ashermann syndrome.

→ If no delivery of placenta >30 min – MRP under General anesthesia

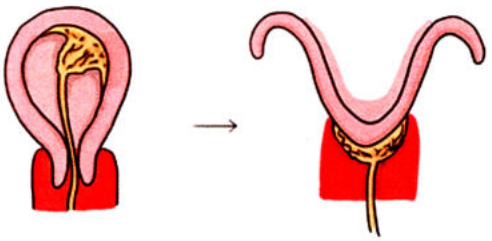
Inversion of uterus

→ If the placenta is pulled with force without giving counter traction

→ Acute Inversion of Placenta occurs.

→ Hemorrhagic shock – M/C/C of death

→ Neurogenic shock



Mx:- Reposition of uterus Manually or Hydrostatic Method

Note:-

→ Last part goes first inside

→ Reposition is done under generally Anesthesia. Reposition is done under general Anesthesia/Terbutaline

→ Once Reposition is done Oxytocin is administered

Note → Sepration of placenta takes place at "Nitabuch layer"/ fibrinoid layer – In case this layer is Absent -

Morbidly Adhered placenta / Placenta Accreta.

→ If placenta Invades the muscular layer → **Placenta Increta**

→ If placenta goes through uterine Muscle and found on Serosa →**Placenta Percreta**

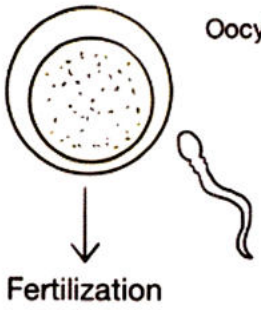
→ M/C/C of placenta Accreta/ Increta/ Percreta → **Low lying placenta / placenta previa**. Other causes:-

- LSCS
- Previous Repeated Currettage
- Infections

Mx of placenta Accreta / Increta/ Pancreta

→ M/C treatment – Obstetric Hysterectomy

→ If bleeding is controlled by sutures, it is followed up by Post – op Methotrexate or Actinomycin is given to degenerate Trophoblast that can cause Trophoblastic diseases / Tumours.



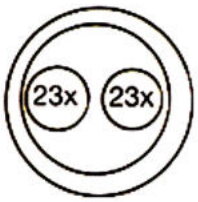
Oocyte (No nulceus- Genetic constituents are dispersed)

Sperm (Have nucleus)

Fertilization

Zygote

23 X from



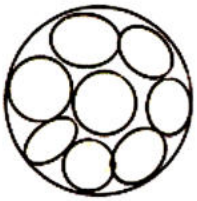
23 X/ Y from

46 XX

2 cell Stage

4 cell stage

8 cell stage



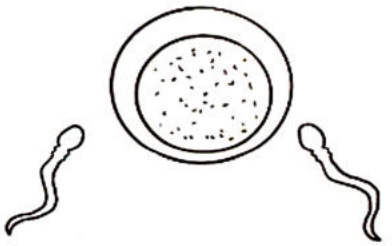
→ In IVF, day 3 Embryo are used. Generally 2-3 embryo are taken and transferred.

Inner cell mass → Fetus

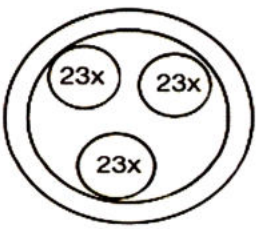
→ Blastocyst- day 5

TrophoEctoderm → Placenta

<u>If Oocyte is fertilised by 2 sperms</u>



(Triploidy)



69 XXX

Partial Mole

Partial Mole

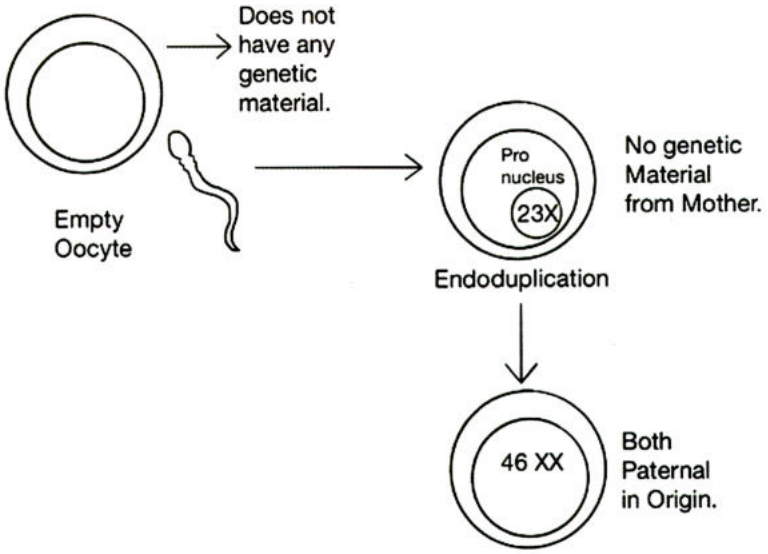
→ Fetus that is partly degenerated by Vesicles.

→ It causes Choriocarcinoma in only 2-3% cases (Almost not seen).

→ Better prognosis than Complete Mole.

→ It can also be 69XXY, 69 XYY, and can never be 69 YYY.

Complete mole



- → It can be 46XX (mainly), 46 XY and can never be 46 YY.
- → The degenerated villi formation occurs → Start imbibing fluid and numerous vesicles appear
- → There is 20% risk of Choriocarcinoma.

Difference between Partial and Complete Mole

	Complete mole	Partial mole
Pathology		
→ Fetal or embryo tissue	→ Absent	→ Present
→ Hydropic swellin of villi	→ Diffuse	→ Focal
→ Trophoblastic hyperplasia	→ Diffuse	→ Focal
→ Implantationsite trophoblast	→ Marked atypia	→ Mild atypia
Karyotype	→ 46XX (mainly)	→ Triploid
	→ 46XY	

Presentation of Complete Mole and Partial Mole

Complete mole:-

→ M/C – bleeding
→ ↑ ↑ BP
→ ↑ ↑TSH (HCG similar to TSH) – Hyperthyroidism
→ Rarely passage of grape like vesicles
→ Uterine size >POG
→ Hyperemesis (due to HCG ↑)

Mx:- Any size – Suction/ Evacuation, followed by check curettage by D/C because there might be retained bits.

Dx → Chest X-ray because it is M/C site of Mets
→ HCG values should be followed for atleast 6 monthsafter it comes negative.
- HCG comes negative in 9 weeks – Complete Mole
- HCG comes negative in 7 weeks – Partial Mole

Note – In follow up period of 6 months, there should not be any pregnancy and we give contraception.

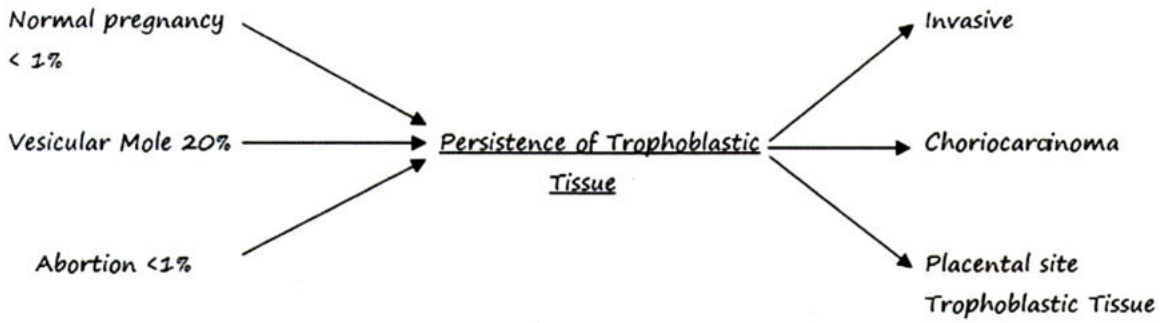
Persistence of Trophoblastic tissue is confirmed by:

→ Continuous bleeding occurs
→ High HCG >100000
→ Persistently ↑ ↑ HCG ± 10% of previous value
→ Bulky / Enlarged uterus

If there is confusion at HPE of either Molar/ Complete Mole or Partial Mole → P_{57} K1P-2 immuno staining is done, and this P_{57} is immune staining only seen with maternal Contribution in the karyotype. Complete mole not show P_{57} K1P-2 staining.

High Risk of choriocarcinoma is seen with:-

→ HCG > 10^5
→ Very large Uterus
→ Theca Lutein cysts (↑ HCG) >6 cm size



Invasive Mole

→ Does not follow normal pregnancy
→ Marker – HCG
→ Surgery is Mainstay of treatment.

Chorio-carcinoma

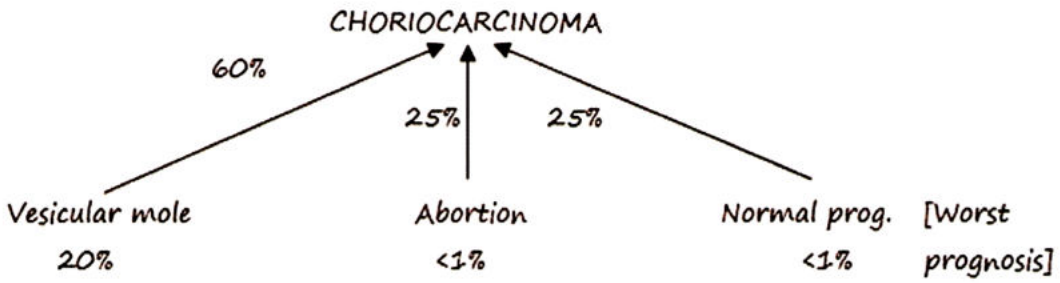
→ Marker – HCG
→ Chemotherapy is treatment of choice
→ For older women hysterectomy can be done

Placental site trophoblastic tumor

→ Marker – HPL

→ Hysterectomy is best management

Choriocarcinoma



Risk factors of choriocarcinoma

Risk factor	0	1	2	4
Age	= 39	> 39		
Antecedent pregnancy	Hydatidiform Mole	Abortion	Term	
Interval (months) from antecedent pregnancy	< 4	4 to 6	7 to 12	> 12
Human Chorionic gonadotrophin (HCG) (IU/L)*	$<10^3$	-10^4	-10^5	$> 10^5$
ABO blood group (female x male)		O x A A x O	AB	B
Largest tumor mass, including uterine(cm)		3-5	>5	
Site of Metastases		Spleen	GI tract	Brain
Number of metastases		1-4	5 - 8	> 8
Prior chemotherapy			Single drug	Two or more

→ Low Risk score <7

→ High Risk score >8

FIGO SCORING

FIGO SCORING	0	1	2	4
Age (years)	<40	≥ 40	-	-
Antecedent pregnancy	Mole	Abortion	Term	
Interval months from end of index pregnancy to treatment	<4	4 - <7	7 - <13	≥ 13
Pretreatment serum hCG (iu/l)	$<10^3$	10^3 - $<10^4$	10^4 - $<10^5$	$\geq 10^5$
Largest tumour size, including uterus (cm)	<3	3 - <5	≥ 5	-
Site of metastases	Lung	Spleen, kidney	Gastro -intestinal	Liver, brain
Number of metastases	-	1-4	5-8	>8
Previous failed chemotherapy	-	-	Single drug	2 or more drugs

Staging of Trophoblastic Disease (choriocarcinoma)

Stage	Site of Metastasis	Prognosis	Management
I	Uterus	Good	Single Agent Chemo
II	Pelvis	Based on scoring	If Low score– single agent chemo
III	Lung	Based on scoring	If High score– Multi agent chem
IV	Distant Metastasi	Poor	Multiagent chemo is used

Note:-

→ Mc. Site of metastasis is Lungs.

→ 2nd MC site of metastasis = vagina (sub urethral nodule) - This appears as a bluish spot in the vaginal area and if you take biopsy of this area, severe hemorrhage can occur, which can not be controlled by suturing & urgent Radiotherapy needs to be given to control it.

Choice of contraception in various situations

→ For Vesicular Mole, contraception needs to be given - Best choice is COCP and IUCD is C/I as it can causes perforation.

→ For Heart ds patient → IUCD here COCP is C/I as they cause fluid retention

→ For Dm patient – Both IUCD & COCP are safe

→ For uncontrolled DM patients – Barrier with spermicidal jelly

→ For STD prevention – Barriers (Double barrier is controversial)

→ Newly Married Couple: COCP

→ Couples staying in separate cities: IUCD

→ Lactational Amenorrhoea – POP

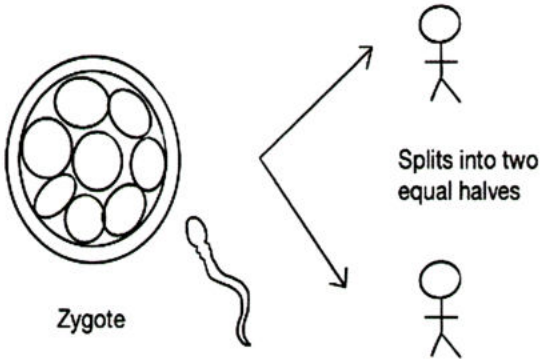
→ Postnatal period >6 wks – IUCD

→ Post placental IUCD (IUCD placed immediately after placental expulsion) - failure rate is 12% acc to WHO.

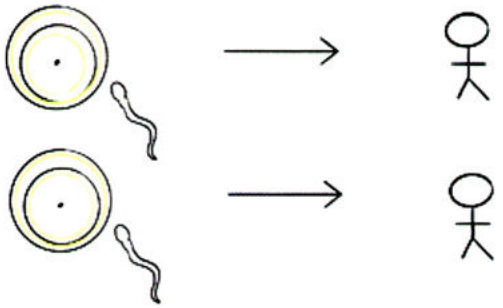
→ Sickle cell Anemia – POP

Twins

Monozygotic Twin



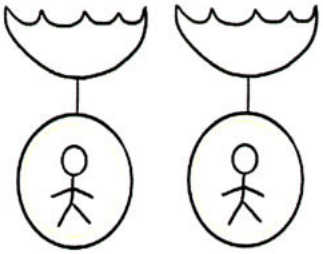
Dizygotic Twin



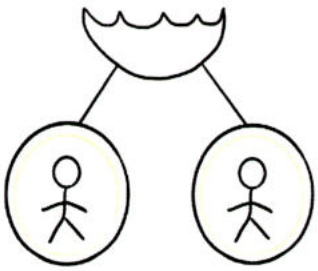
Two separate Oocytes fertilised by separate sperms.

Monozygotic Pregnancy

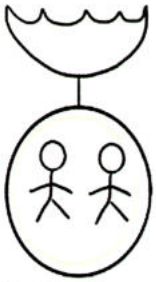
If split is < 3 days (30%)-
Dichorionic Diamniotic



If split is in 3-8 days (40% commonest)-
Monochorionic Diamniotic



If split is after 8 days then (8-12 days)-
Monochorionic Monoamniotic (Momo twins)



If split is > 12 days-
Conjoined Twins
(because embryonic plate is already formed).

→ **Monozygotic Twin**→ Identical Twins and 1/250 (less common)

→ **Dizygotic Twin** → Not Identical so Fraternal Twins, always Dichorionic Diamnioticand 1/60-80 (More common).

Note : Dichorionic Diamniotic pregnancy can also be seen in monozygotic twins if split happens before 3 days as seen above.

Complications In Twin Pregnancy

→ In case of Monochorionic Pregnancy there is sharing of blood between the two babies via deep artero-venous anastomosis

- The receiving baby – Plethoric High Hb More weight
- The giving baby – Thin & weak, Anemic

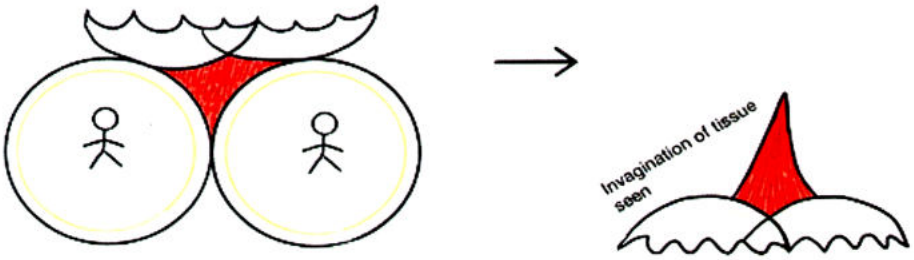
→ In case of Monoamniotic Pregnancy:

- Cord Accidents
- Abruption
- Single baby demise leading to DIC
- Premature Rupture of membranes
- Preterm Labor

Diagnosis Of Twin Pregnancy

→ USG – Done At 12th week of Pregnancy to find out chorionicity - Chorionicity scan

This is Twin Peak Sign



2 chorion arising from two separate placenta

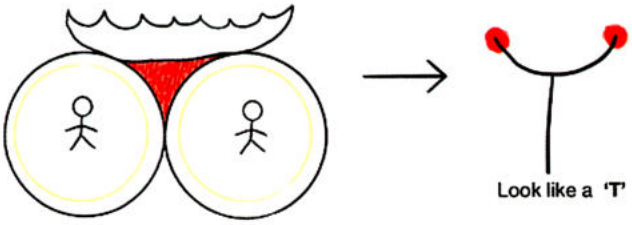
Looks like a peak

Seen in Dizygotic Dichorionic Pregnancy

→ But it still does not tell about the Zygocity of Pregnancy ie whether it is dizygotic or Monozygotic

→ If there is no invagination of tissue seen

If there is no invagination of tissue seen



Look like a 'T'

This is 'T' sign seen in Monochorionic Diamniotic Pregnancy.

Emergency Contraception

→ Contraception given to prevent pregnancy, within 72 hours of unprotected intercourse.

→ Abortion is not a method of emergency contraception

FSH → Estrogen

LS → Progesterone

→ Combined oral contraceptive pills (COCs)→ Estrogen + Progesterone

↓

Negative feedback to pituitary (No FSH and LH to act on ovary)

↓

Supresses Estrogen+Progesterone formation by ovary.

→ COC's cause artificial menstruation in the female.
- Cycles are regular, anovulatory

→ Dose of Estrogen is low in COC (Max – 0.03mg), so it
- Reduces bleeding
- Control anemia
- Since Estrogen is less – Ovarian Cancers, endometrial cancers and fibroids are less.

For Emergency contraception, best given is –

Levonogesterol (LNG)-0.75mgx2 tablets - 12 hours apart or 1.5mgx1Tab. MOA:-
→ ↓ Implantation by making endometrium too fluffy or hypersecretory.
→ ↓ tubal Motility
→ ↓ Ovulation (by suppressing LH surge)

COCP- Yuzpee regime (outdated) - 2Tab (morning) 100µg - 12 hrs apart 2nd tab (evening)-100µg.
MOA - ↓ ovulation
- ↓ Implantation

Mifepristone: Ru 486-Dose – 25-50 mg; MOA:-
→ Antiprogestin
→ ↓ implantation

IUCD

→ Effective upto 5 days of unprotected intercourse
→ Most effective method but not DOC

Note:- Drugs of Emergency contraception are not prescription drugs,these are "OVER THE COUNTER" drugs

Ullipristal Acetate
→ Selective Progesterone receptor modulator (SPRM)
→ Equally effective upto 5 days of unprotected intercourse.

1st generation → Lippes loop
2nd generation → Cu7, Cu 380 Au/Ag → Effective for 5 years (380 - Surface area of cu in mm^2 on device).
→ Paragard – Effective upto 10 years

3rd generation- Progesterone Containing devices:-

→ Progestasert – 38mg, releasing 65mcg/day

→ LNG devices(MIRENA) – 52mg, releasing 20µg /day

Also used for Management of:-

- Menorrhagia
- Endometriosis
- Endometrial hyperplasia

MOA of IUCD:-

→ Foreign body Action

→ Endometrium non receptive

→ ↓ ↓ Ovulation

Note: Progestasert having above actions; also act by thickening of cervical Mucous (main Action). LNG also make cervical mucous thick but main action is making endometrium non-receptive.

Side effects of IUCD

→ M/C – Bleeding > Pain

→ Perforation at Insertion

Note: - For first three months of bleeding after IUCD we give prophylactically NSAIDS and Tranexemic Acid to some patients

Extra:- Scenario 1- IUCD patient got pregnant

Mx = 1st step – Remove IUCD, If thread are not visible then Continue pregnancy (50% chances of Abortion) Or MTP is done.

Note:- Overall chances of ectopic pregnancy is more with patient having IUCD as compare to Normal Women having pregnancy.

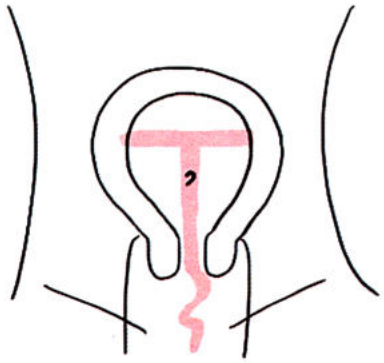
Scenario 2 Lost device/IUCD.

Mx:- First step - Exploration in OPD with Artery Forceps, if not found then do USG and locate device.

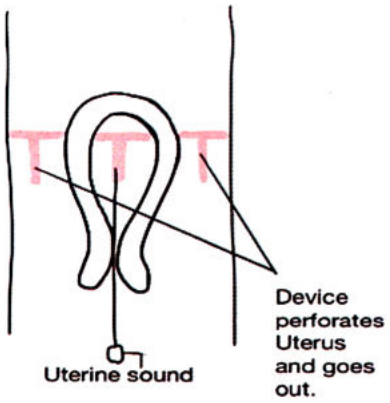
↓ if not found

Radiography (X-Ray) – Rule out pregnancy first beforehand.

AP view



Lateral view



→ In lateral view if the Uterine sound and Copper device are in same plane → Copper T is inside

Sterlization

Vasectomy - failure rate <0.1 %

Non scalpel vasectomy – done by Artery forceps under local Anesthesia

→ Avoid Intercourse without condoms/Barriers for 3 months or till 30 ejaculations.

→ After 3 months – semen Analysis is done – to see Azoospermia.

Note-Reversibility is only 30-35% successful.

Tubectomy

Puerperal Tubectomy- Within first 7-10 days – (Best done 2-3 days)

- Done by Open technique – Mini laprotomy (1.5 – 2inches)

Interval sterlisation→ after 6 weeks → Mostly done by laproscopy

Concurrent sterlisation → done along with another Method like LSCS, MTP

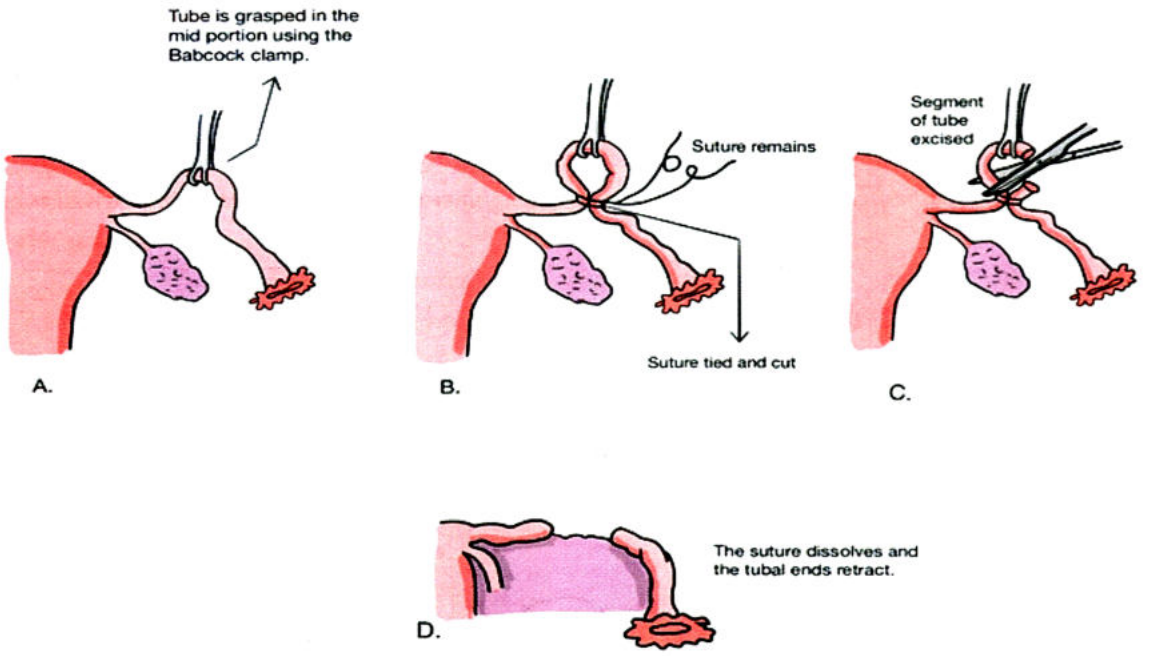
Reanastomosis of Tubectomy (Reversibility)

→ Isthemo Isthemic Reanastomosis – Upto 80% successful

→ Vaso vasal reanastomosis - upto 35%

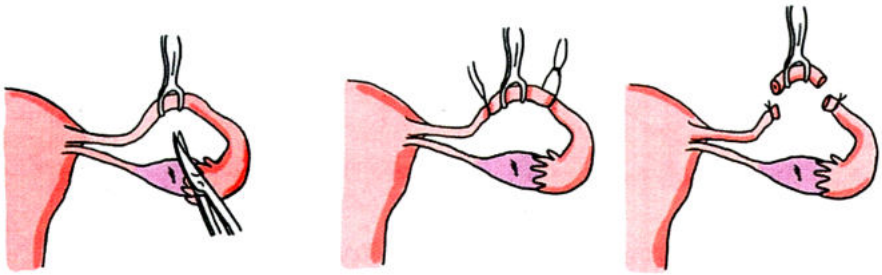
→ Isthemo – Ampullary Reanastomosis – 30% successful

M/C method for tubectomy- Modified Pomeroy's method → Risk of spontaneous Reanastomosis

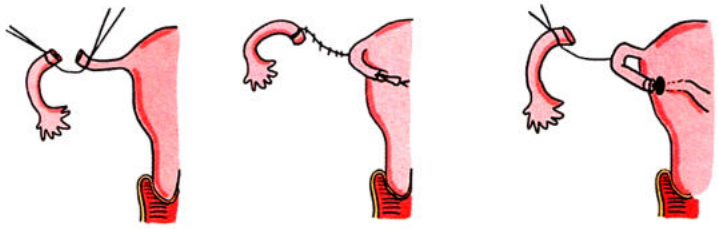


Other methods

→ **Parkland's Technique**



→ **Irwing Sx Technique:** - We cut the tube and buryin uterus and the other end buried into mesosalpinx



KRONER'S Sx: - Fimbriectomy

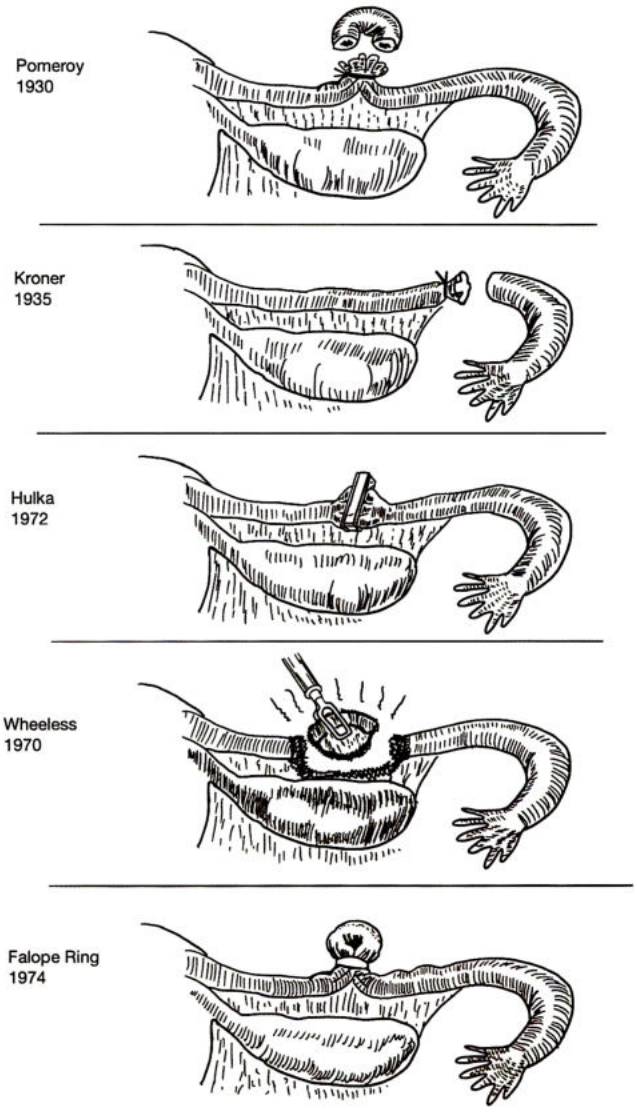
Madlener's Method: - Only crushing of tube done – no cutting, so high failure rate, so not done.

Essure ring

- Hysteroscopic procedure and putting of Nitinol (Nickel + Titanium) Coil into tube – Causes fibrosis of the Interstitial part of tube within 3 months.
- Hysterosalpingography is done after 3 months to confirm the fibrosis

Extra:-

→ Methods of tubectomy:-



→ Failure rate of contraception drugs is calculated by PEARL INDEX –total number of accidental pregnancies x 1200.

→ Emergency Contraception – levonorgesteral – 1.5 mg 1 single dose MOC is other than IUCD.

→ Best method - Permanent methods – vasectomy/tubectomy.

→ Rates of failures :

- Vasectomy – 0.1%
- Tubectomy – 0.2%
- IUCDs 0.5 -0.6 % (Copper devices) and 0.2% (Levonorgesterone)
- Combined OCPs – 0.6 – 0.7%
- Preg. Only pill – 1.2%
- Barrier methods - ♂ and ♀- 14-20%
- Natural method – X
- IMPLANTS – Norplant, Implenon - 0.05%

Progesterone Implants

Norplant – 6 stems – 36 mg each = 216 mm of LNG, applied as subdermal implant over the arm and effective till 5 yrs.

Norplant – 2 "JADELLE" - 75mg x2 = 150 mg (2 stems) of LNG.

Etonorgestrel Implants -Synthetic /degradation part of dezogesterel and extremely effective.

Implanon: 68mg single stem effective till 3 yrs withdrawn in most countries

Nexplanon: Ba Coated – radio opaque Implant so easy to locate & remove later on

COCPs → E + P → MOA – Inhibit FSH and LH from pituitary

↓

Ovary suppressed

- Estrogen causes proliferation of uterus
- Progesterone cause secretion of endometrium
- So women will have artificial periods
- Women do not ovulate & do not conceive

→ MALA-D, MALA-N

- Estrogen = 0.03 mg = 30 µg
- Progesterone = 0.15 mg = 150 µg

Natural methods of contraception - 60 failures / 100 WY

→ Calendar / Rhythm / Safe period method

- From 11th – 16th day – high chance of conception as on 14th day ovulation occurs
- So avoid sex in this period.

→ Cervical Mucus method

- Watery mucus occurs during ovulation period
- Causes wetness in vagina
- Avoid intercourse in this period
- Known as Billing's method

→ Withdrawal Method

→ Basal Body temp Increases during ovulation

- Progesterone hormone is thermogenic hormone
- Rise of 0.5 F in body temp.

Staging

Stage 1:

The carcinoma is strictly confined to the cervix uteri (extension to the corpus should be disregarded)

- **IA** Invasive carcinoma that can be diagnosed only by microscopy, with maximum depth of invasion < 5 mm
    - **IA1** Measured stromal invasion<3mm in depth
    - **IA2** Measured stromal invasion ≥ 3mm and <5mm in depth
- **IB** Invasive carcinoma with measured deepest invasion ≥5 mm (greater than stage 1A), lesion limited to the cervix uteri
    - **IB1** Invasive carcinoma ≥5mm depth of stromal invasion of <2 cm in greatest dimension
    - **IB2** Invasive carcinoma ≥2 cm and <4 cm in greatest dimension
    - **IB3** Invasive carcinoma ≥4 cm in greatest dimension

New Changes

1A = Microscopic cancer; Transverse spread is not included (earlier classification used transverse spread as well)

1B = clinically obvious

→ Now 1B is further divided into 1B1, 1B2, 1B3

Stage II

The carcinoma invades beyond the uterus, but has not extended onto the lower third of the vagina or to the pelvic wall

- **IIA** Involvement limited to the upper two-thirds of the vagina without parametrial involvement
    - **IIA1** Invasive carcinoma <4 cm in greatest dimension
    - **IIA2** Invasive carcinoma ≥4 cm in greatest dimension
- **IIB** with parametrial involvement but not up to the pelvic wall

Stage III:

The carcinoma involves the lower third of the vagina and/or extends to the pelvic wall and/or causes hydronephrosis or non-functioning kidney and/or involves pelvic and/or paraaortic lymph nodes

- **IIIA** Carcinoma involves the lower third of the vagina, with no extension to the pelvic wall
- **IIIB** Extension to the pelvic wall and/ or hydronephrosis or non-functioning kidney (unless known to be due to another cause)
- **IIIC** Involvement of pelvic and/or paraaortic lymph nodes, irrespective of tumor size and extent (with r and p notations)
    - **IIIC1** Pelvic lymph node metastasis only
    - **IIIC2** Paraaortic lymph node metastasis

Tips to remember:

IIA → upper 2/3rd of vagina
IIIA → Lower 1/3rd of vagina

IIB = Parametrial involvement short of pelvic wall
IIIB = Parametrial involvement till pelvic side wall

→ Full growth from cervix till the pelvic side wall (No cancer free area in between)

Whole area of parametrium is involved, the ureters get compressed in between Leads to

↓

Hydronephrosis

Stage IIIC: New development in staging of carcinoma cervix

→ All cancers in gynaecology are staged surgically

→ Cervical cancer staged clinically → on per vaginal & per rectal examination

Till September 2018

→ Imaging was not required in staging of carcinoma cervix

→ From September 2018, imaging is required for staging to look for paraaortic or Pelvic Lymph nodes involvement

- Method of imaging can be
  - USG
  - MRI
  - CT
  - PET - CT
  - MRI - PET

Stage IV:

The carcinoma has extended beyond the true pelvis or has involved (biopsy proven) the mucosa of the bladder or rectum. A bullous edema, as such, does not permit a case to be allotted to stage IV

- **IVA** Spread of the growth to adjacent organs
- **IVB** Spread to distant organs

Epidemiology

M/C cancer in female genitalia in India = Carcinoma cervix

M/C Cancer in females overall = Carcinoma breast).

- M/C associated virus is HPV 16
- Most invasive is HPV 18

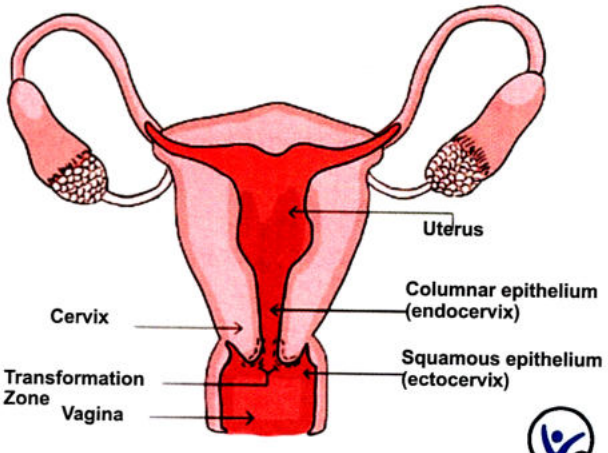
Screening for carcinoma cervix

→ Time to start pap smear screening: 3 years after first exposure to sexual activity (In western countries, it is started at 21 years of age)

→ Frequency: Yearly
  - If Pap smear negative and HPV DNA negative, repeat Pap smear after 3 years.

→ When to stop screening
  - >65 years of age (earlier it was 70 years)
  - If 10 smears negative after menopause

→ Site for PAP smear: Transformation zone/Squamo-columnar junction First site which gets affected by HPV



Dr. Vivek Nalgirkar

Results of PAP smear

- **CIN I** : Less than 1/3rd cells abnormal
- **CIN II**: 1/3rd to 2/3rd cells abnormal
- **CIN III**: >2/3rd cells abnormal
- **Carcinoma in situ** :All cells on smear are abnormal (atypical)

CIN I $\xrightarrow{5\text{ years}}$ CIN III $\xrightarrow{10\text{ years}}$ carcinoma cervix

In **CIN I**: More frequent PAP smears and typing of HPV DNA (High risk or low risk)

CIN III or Post coital bleeding or carcinoma in situ

↓

Colposcopy directed biopsy

CIN III Invasion of abnormal cells into stroma

↓

: LEEP (Loop electrosurgical Excision Procedure)

: LASER – Expensive

: >40years – cone excision (preserves uterine function) or

– Hysterectomy

LEEP Earlier known as LLETZ (Large loop excision of Transformation zone)

Invasion into stroma

↓

Invasive Carcinoma Cervix

↓

T/t: Radical hysterectomy

- Stage till IIA → Radical hysterectomy
- Beyond IIB → Radiotherapy + Chemotherapy (Chemo Radiation)

In India most patients present in advanced stages → on chemoradiation 5-year survival rate is atleast 50%.

CARCINOMA ENDOMETERIUM

FIGO stage	Description
Stage IA	Tumor confined to the uterus, no invasion or invasion or invasion of less than one-half of the myometrial thickness
Stage IB	Tumor confined to the uterus with invasion of more than one-half of the myometrial thickness
Stage II	The tumor invades the cervical stroma, but does not extend beyond the uterus
Stage IIIA	The tumor invades the uterine serosa or adnexa
Stage IIIB	Vaginal and/or parametrial involvement
Stage IIIC	The tumor has spread to pelvic or para-aortic lymph nodes
Stage IIIC1	Pelvic lymph node involvement
Stage IIIC2	Para-aortic lymph node involvement (with or without pelvic nodes)
Stage IVA	Tumor invasion of the bladder and/or bowel mucosa
Stage IVB	Distant metastases including abdominal metastases and/or inguinal lymph nodes

Imp: Inguinal lymph node involvement (stage IV B)

→ Round ligaments which go from uterus take lymphatic along inguinal canal and drain into inguinal group of lymph nodes.

Epidemiology

- CA endometrium is a cancer of well to do females (cancer of rich & famous)
- Obese
- Hypertensive
- Diabetic

→ Carcinoma Cervix is more likely in Indian population- typically a thin, multiparous female.

Risk factors

- ↑ Estrogen
- Obesity – HTN - DM (Corpus Cancer syndrome)
- Nulliparous
- HRT
- Tamoxifen therapy (SERM) [Estrogenic effect on endometrium .Suppress the hormonal function of Tamoxifen by GnRH analogue]
- Familial Predisposition is seen in Ca. Breast, Ca. Endometrium and Ca. ovary. First degree relatives can have any of these.

→ M/C carcinoma endometrium: **Adenocarcinoma**

→ Prognosis: Most important Prognostic feature is : **staging**

Other important factors for prognosis are: -

→ Grading
→ ER status
→ Age
→ Prior Treatment
→ LN status
→ Chemotherapy

CARCINOMA OVARY

STAGE I: Tumor confined to ovaries	
IA	Tumor limited to 1 ovary, capsule intact, no tumor on surface, negative washings
IB	Tumor involves both ovaries otherwise like IA
IC: Tumor limited to 1 or both ovaries	
IC1	Surgical spill
IC2	Capsule rupture before surgery or tumor on ovarian surface
IC3	Malignant cells in the ascites or peritoneal washings

STAGE II: Tumor involves 1 or both ovaries with pelvic extension (below the pelvic brim) or primary peritoneal cancer	
IIA	Extension and/or implant on uterus and/or fallopian tubes
IIB	Extension to other pelvic intraperitoneal tissues

STAGE III: Tumor involves 1 or both ovaries with cytologically or histologically confirmed spread to the peritoneum outside the pelvis and/or metastasis to the retroperitoneal lymph nodes		
IIIA: Positive retroperitoneal lymph nodes and/or microscopic metastasis beyond the pelvis)		
IIIA1	Positive retroperitoneal lymph nodes only	
	IIIA 1 (i) IIIA 1 (ii)	Metastasis < 10 mm Metastasis > 10 mm
IIIA2	Microscopic, extrapelvic (above the brim) peritoneal involvement + positive retroperitoneal lymph nodes	
IIIB	Macroscopic, extrapelvic, peritoneal metastasis < 2 cm + Positive retroperitoneal lymph nodes. Include extension to capsule of liver/ spleen	
IIIC	Macroscopic, extrapelvic, peritoneal metastasis > 2 cm + positive retroperitoneal lymph nodes. Includes extension to capsule of liver/ spleen	

STAGE IV: Distant metastasis excluding peritoneal metastasis	
IVA	Pleural effusion with positive cytology
IVB	Hepatic and/or splenic parenchymal metastasis, metastasis to extra abdominal organs (including inguinal lymph nodes and lymph nodes outside of the abdominal cavity)

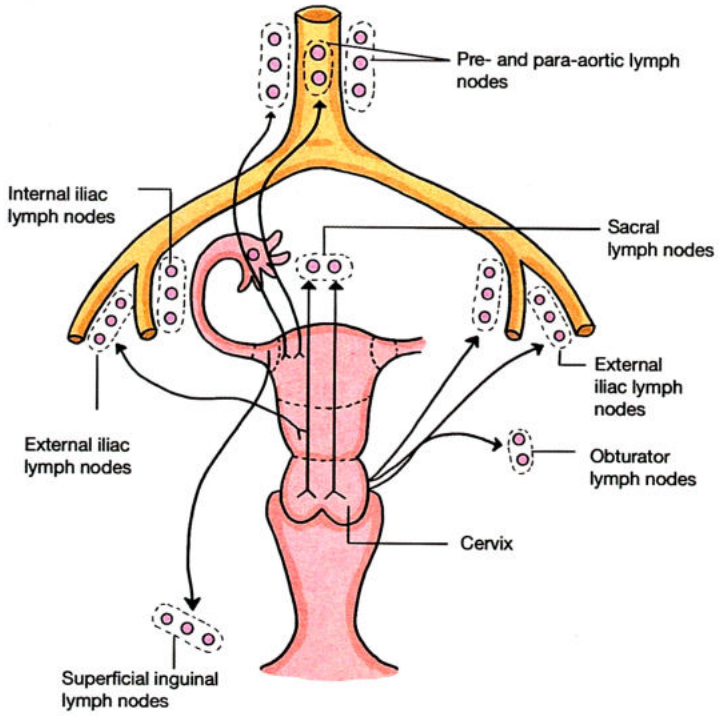
→ Ca ovary = Too much ovulation → Too much epithelial injury → In nulliparous women (they do not have breaks of pregnancy; keep ovulating)

Classification of carcinoma ovary modified in 2014 (FIGO)

New changes in 2014

- → Stage IC: IC1 (Surgical spill), IC2, IC3 now changes
- → Positive retroperitoneal lymph node involvement is III A1
- → Inguinal lymph nodes involvement is IV B
- → IV A: Stage newly formed i.e. pleural effusion with positive cytology

Comparison of Ca. ovary, cervix and endometrium



Inguinal lymph nodes involvement

- Cervix does not drain into inguinal group of lymph nodes.
- Inguinal lymph nodes involvement in ovary and endometrium is **stage IVB**

Pelvic & paraaortic lymph node involvement

- Ovarian cancer: **Stage IIB** (Extension to other pelvic intraperitoneal tissues – pelvis, rectum, bladder.
- Ca. cervix and Ca. Endometrium: **Stage IIIC1 and IIIC2**

Epidemiology

M/C reason of ovary cancer: Epithelium Injured by ovulation every month.

→ Ca. ovary = Too much ovulation → Too much epithelial injury →

nulliparous women (they do not have breaks of pregnancy; keep ovulating)

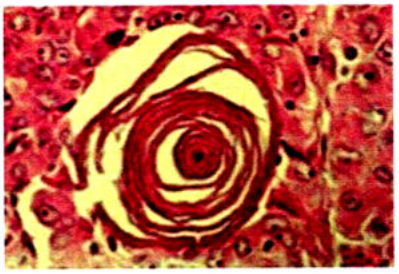
Types of Ovarian Cancer

Epithelial cancer (70%) (M/C type)

Bilateral cancer, in older women

- **Serous**
  - M/C epithelial cancer of ovary
  - Large, unilocular, cystic tumor
  - Psammoma bodies.

Serous tumour



Psammoma bodies

- **Mucinous**
  - Large, multiloculated, cystic tumor
  - Pseudomyxoma peritonei

↓

Rupture of mucinous cyst→ mucin gets deposited in abdomen → Mucinous ascites

- **Brenner tumor** (M/C cause of pseudo meig's syndrome)
  - Derived from transitional epithelium, no secretions
  - Benign, post-menopausal bleeding

Imp point: Meigs syndrome: Fibroma + ascites + Pleural effusion

Pseudo Meigs syndrome: Any other ovarian tumor + ascites + Pleural effusion)

- Ovarian tumors develop from injured epithelium
- Ovarian epithelium – totipotent (can become like any epithelium of body)
- Mostly converts into epithelium of pelvic tissues

E.g.:1. Fallopian tube – Serous cystadenoma

2. Cervix – Mucinous cystadenoma
3. Bladder – Transitional cell type of tumor – Brenner's tumor
4. Endometrium = endometroid tumor (6-8% of ovarian tumors) ,Includes endometriosis related tumors

Borderline epithelial ovarian tumors:

features
- Neoplasia, Atypia
- Epithelial proliferation
- No destructive stromal invasion
- Low malignant potential tumor
- Surgery alone is adequate treatment.
- Good prognosis tumor.

Germ cell Tumors

- Unilateral
- In younger women
- M/C: Teratoma → M/C Teratoma is Dermoid

Dermoid

- 90% of Teratomas, benign
- M/C tumor of pregnancy, Torsion
- B/L in 15% cases
- Have all three germ layers – ectoderm, mesoderm, endoderm
- Have bone, teeth, hair, cartilage, sebaceous material

Dysgerminoma

- M/C germ cell malignancy (40-45%)
- Associated with Hypercalcemia
- 5% Seen along with Dysgenic gonads
- B/L in 10-15% of cases
- Only radiosensitive tumor of ovary
- ↑ LDH, ↑ Placental Alkaline Phosphatase
- No increase in α - feto protein (AFP)

Yolk sac tumor/ endodermal sinus tumor - ↑AFP
-Specific: α-1 antitrypsin

Embryonal tumor - ↑ AFP
- Specific: HCG

Sex - Cord tumors

Granulosa cell tumor

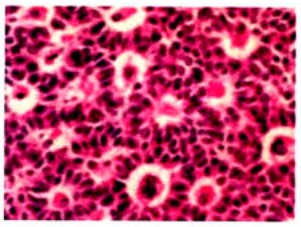
Estrogen Producing

Effects of excess estrogen:

→ In young girl (6-7 years old) – Precocious Puberty

→ Middle aged women – Too much estrogen → Too much endometrium → Menorrhagia → Hyperplasia of uterus and endometrial cancer later on

- **Call Exner bodies:** rosettes of granulosa cells with an eosinophilic center
- Marker is **Inhibin**



Call Exner bodies

Sertoli- leydig tumors

- In women, produce ↑ androgens
- C/F : Rapid onset hirsutism (any age)
  : Oligomenorrhea/ Amenorrhea

Krukenberg Tumour

M/C secondary to ovary is from: Ca. stomach (mostly), Ca. breast

- B/L tumor, large in size
- Has signet ring cells.
- Firm to solid tumor
- May have cystic changes
- Known as Krukenberg tumor

Non-Neoplastic ovarian cyst

- Corpus luteum cyst
- Follicular cyst
- Hemorrhagic cyst
- Theca lutein cyst

Conservative Treatment

- Theca lutein cyst occurs due to ↑ hCG in molar pregnancy, twin
- Can be upto 5cm, but mostly resolve spontaneously in 3-4 months
- Follow up for few months

On USG: Signs of malignancy

- B/L
- Surface growth
- Cystic and solid appearance
- Ascites
- Thick septate tumor
- Deposits in P.O.D. (Pouch of Douglas)
- LN seen

Imp. Points:

- Most non-neoplastic ovarian cysts are self-limiting
- Theca lutein cyst: T/t→ Remove predisposing cause (molar pregnancy)
- In case of twin, pregnancy after, delivery cyst vanishes by itself.

Carcinoma Vulva

Stage I	Tumor confined to the vulva
IA	Lesions ≤ 2 cm in size, confined to the vulva or perineum and with stromal Invasion ≤ 1.0 mm, no nodal metastasis
IB	Lesions >2 cm in size or with stromal invasion >1.0 mm. confined to the vulva or perineum, with negative nodes
Stage II	Tumor of any size with extension to adjacent perineal structures (1/3 lower urethra, 1/3 lower vagina, anus) with negative nodes
Stage III	Tumor of any size with or without extension to adjacent perineal structures (1/3 lower urethra, 1/3 lower vagina, anus) with positive inguino-femoral lymph nodes
IIIA	i. With 1 lymph node metastasis (≥5 mm), or ii. 1~2 lymph node metastasis (<5 mm)
IIIB	i. With 2 or more lymph node metastasis (≥5 mm), or ii. 3 or more lymph node metastasis (<5 mm)
IIIC	With positive nodes with extracapsular spread
Stage IV	Tumor invades other regional (2/3 upper urethra, 2/3 upper vagina), or distant structures
IVA	Tumor invades any of the following: i. Upper urethral and/or vaginal mucosa, bladder mucosa, rectal mucosa, of fixed to pelvic bone, or ii. Fixed or ulcerated inguino-femoral lymph nodes
IVB	Any distant metastasis including pelvic lymph nodes

Remember:

Stage I and II: No lymph node involvement

Stage II: lower part of adjacent organs (1/3 lower urethra, 1/3 lower vagina, anus)

Stage III: Positive inguino femoral lymph nodes

→ In ca. vulva; more the number of lymph nodes – (worse is prognosis) higher is stage

→ A single LN which is of size more than 5 mm is stage III A (I)

→ 1-2 LNs with size < 5 mm is stage III A (ii)

Stage IV A (I): If growth on vulva is stuck to pelvic bone

IV A (ii): Inguino femoral LNs fixed or ulcerated

Epidemiology

- >65 years of age, older women
- Most morbid: Morbidity d/t surgical treatment (40%)
- Limit surgery
- If a small ulcer, stage 1 = simple vulvectomy (if no LN involvement)
- From stage 2, LN involvement assessed
    - May need wide excision based on +/- LN status.

Assessment of LN status

- Inject dye(Technetium 99) into the vulva, if same dye stains LN -confirms involvement of LN (stage 3 tumor)
- Beyond stage 3: wide local excision /Radical vulvectomy + LN excision
- More extensive procedure, more morbidity
- There is cross-over of lymphatics in vulvar region. Vulva does not drain into pelvic LN.
- If pelvic LN are involved = Stage IV

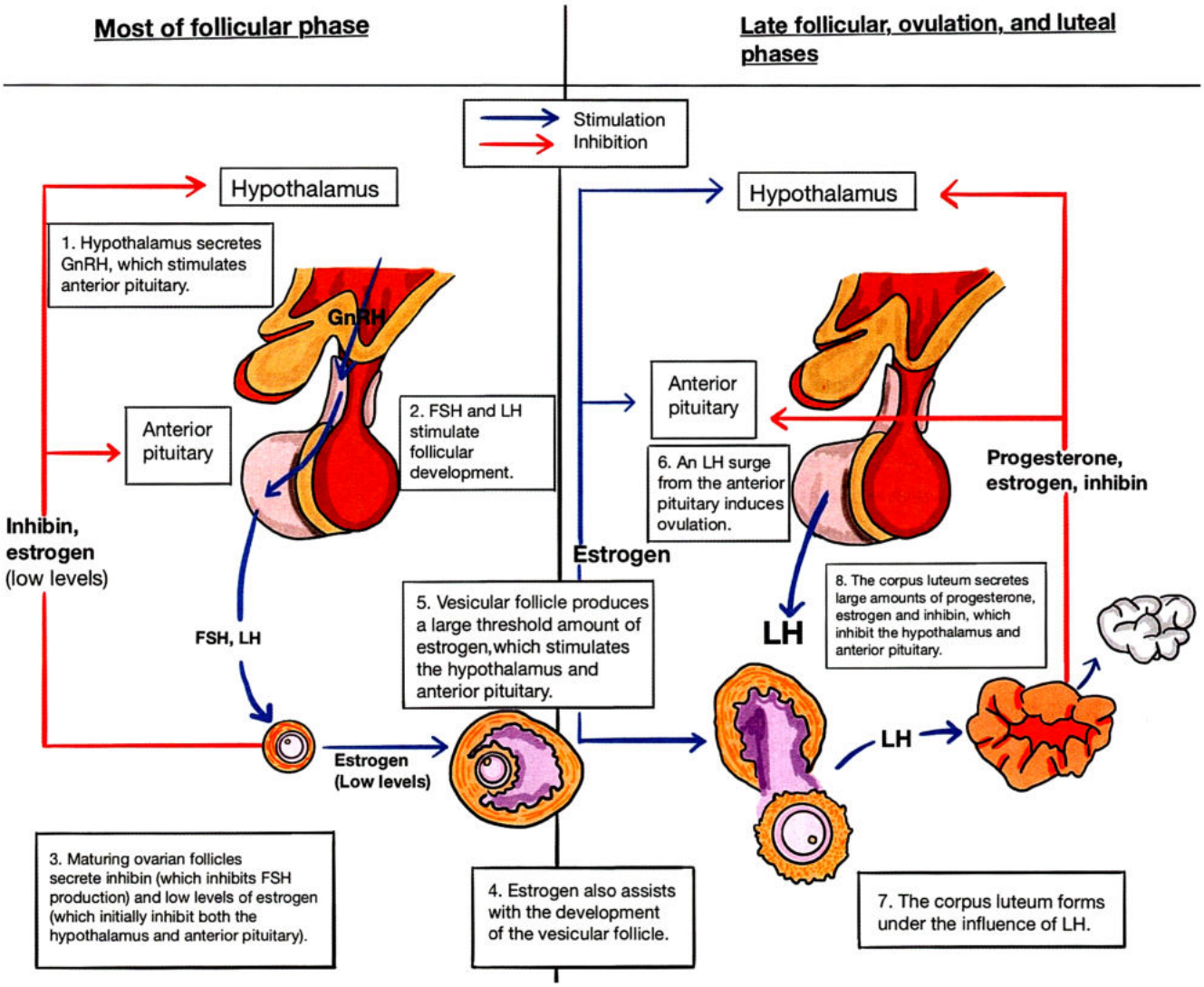
Classical presentation

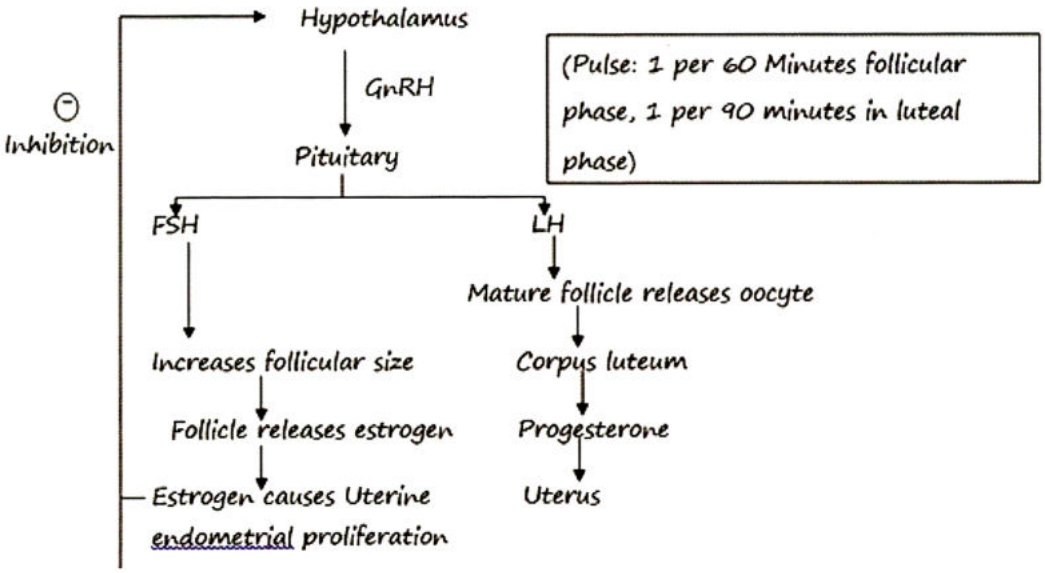
- Older women
- Itching (itch – scratch cycle: itch leads to release of more histamine which further increases itching)
- Not associated with HPV
- Lichen sclerosus (Keratinizing variety)

In younger women:

- HPV associated (HPV 6, 11)
- Smoking
- Multifocal basaloid vulvar cancer

NORMAL MENSTRUAL CYCLE

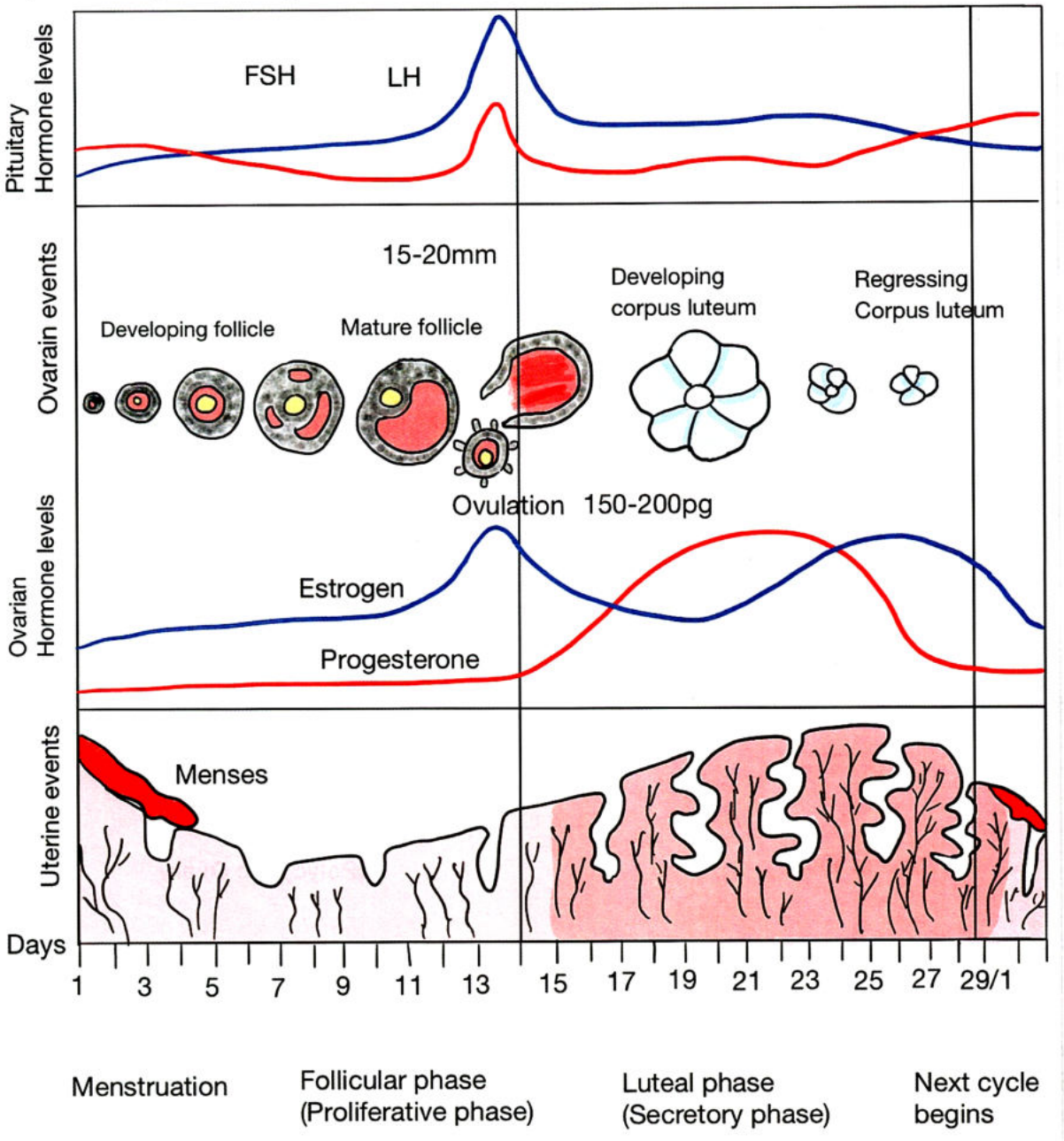




Dr. Vivek Nalgirkar

- In developing follicle androgens converted to estrogen due to enzyme aromatase (produced by theca cells)
- FSH: Purpose of FSH, when it leaves brain, is to give estrogen back to brain
- LH: Purpose of LH, when it leaves the brain, is to ovulate the follicle and make corpus luteum and give progesterone which gives feedback to brain and ↓ LH.

(FSH: Estrogen; LH: Progesterone)



Follicular phase

- Estrogen: Progressive proliferation of glands of endometrium

Secretory phase

- Progesterone: glands of endometrium become secretory and get stabilized
  - Secretary endometrium: helps implantation
  - Stabilized endometrium: Prevents endometrial cancer

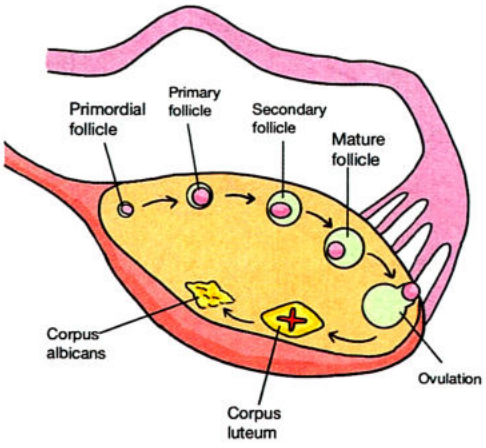
- Anovulatory women: too much endometrial proliferation
  ↓more likely
  Endometrial cancer
- Corpus luteum maximum work **9-10 days**; when it degenerates (degenerates in 15 days) progesterone ↓; bleeding starts

PCOD/PCOS

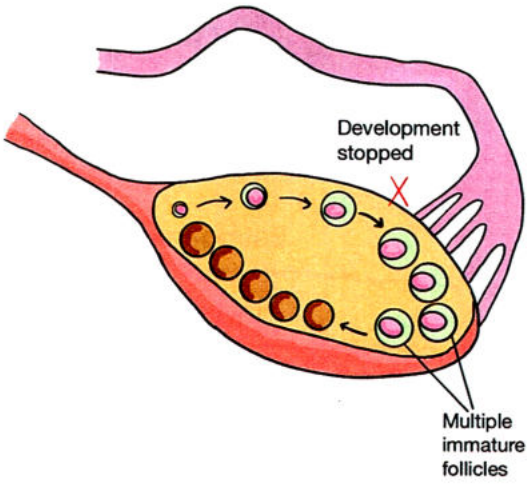
Polycystic ovarian disease/ Syndrome

Normal Ovary → oocyte released → crater formed, gets filled with blood → heals and forms scar
PCOD ovary
Anovulatory condition

- Smooth on surface, sclerotic, pearly white in color
- On cut section: necklace of pearls appearance
- Mildly enlarged ovary (2-5 times of normal ovary)
- Many Small follicles form estrogen
- It does not have cysts



Normal Ovary



Polycystic Ovary

- For ovulation follicle size should be 15-20 mm size
- In PCOS multiple ovarian follicle size (2-6 mm size)
  ↓
  Not mature
- Endometrium sheds in (40, 50, 90 days) when endometrium outgrows its blood supply (blood supply is from basal side),becomes ischemic at top and sheds.

C/F:

- Anovulatory cycles
- Delayed cycles: Oligomenorrhea
- Amenorrhea (>3 months no menstruation)
- Hirsutism and Hyperandrogenism

- Estrogen secreted by multiple follicles → Causes LH Surge

No ovulation → LH excessively increased

LH: FSH (1:1 normal) - increases to 3:1

Ovarian stroma (Thick) LH (↑), Androgens ↑

- Testosterone, ↑ Androstenedione, ↓ Sex Binding Globulin (SBG ↓)

  Causes Hirsutism, Hyperandrogenism

- Why is ovarian follicle size not increasing in PCOS:

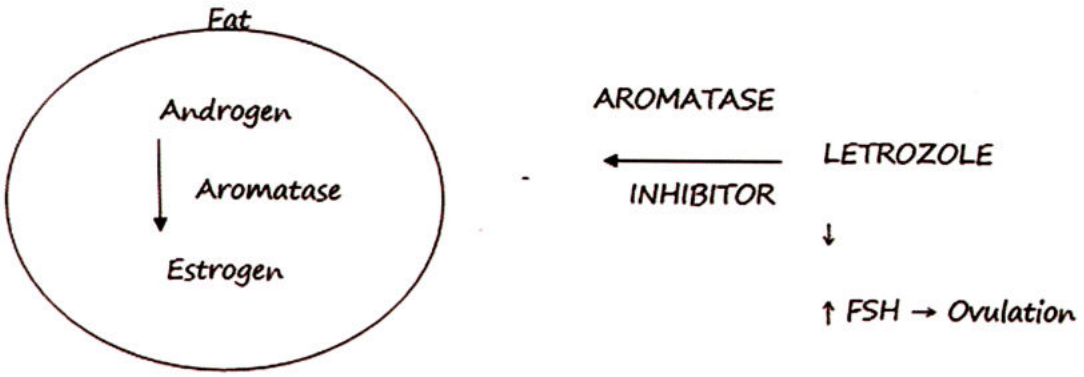
→ Insulin required for carrying glucose moiety from circulation across cell membranes (in skeletal muscles, all tissues, then to mitochondria)

→ In PCOS, there is Insulin resistance; glucose does not enter ovary, so not much energy. This leads to decreased growth of follicles in ovary.

Treatment

→ **Anovulation** 1. Weight reduction ↑ Insulin sensitivity → in 30% females ovulation occurs.

2. Insulin sensitizers: Metformin → 30% females ovulate
3. Hypothalamic estrogen receptor blocker (stops estrogen action on brain and FSH)
4. Clomiphene citrate: 80% ovulate 40% conceive (after Treatment for 3-6 months)
5. Injection of Recombinant FSH
6. Injection of human menopausal Gonadotropin (hMG) 75 IU (contains LH and FSH)



→ Since July 2018, **Letrozole:- DOC for ovulation induction**

- Reasons: Lesser twin rate
  : Lesser complications
  : Lesser teratogenic
  : Safer drug (in relation to effect on endometrium)

Irregular cycles

: Combined OCPs → "Artificial cycles" (No action of pituitary, ovary = no ovulation)

: Low dose pills, 21 days → less periods, Regular periods

: Progesterone Only Pills

Give pills for 10 days, after the day 14 and stop. On day 28-29, endometrium sheds→menstruation

OR

Give 5 pills from day 20-25 and stop. On day 28-29, endometrium sheds-menstruation

→ Regular cycles

15% women have PCOS

Proper management → can lead normal life

- Thin (↓ weight)
- Regular cycles (by using OCPs)
- When fertility needed: OCPs can be stopped

↓ 2-3 years

Can spontaneously ovulate & have pregnancy

→ If no pregnancy then give -

Clomiphene citrate ⟶ Letrozole

↓

Ovulates

Diagnosis of PCOS

RotterDam criteria (2003)/ASRM (American society for Reproductive Medicine)/European society for human reproduction & Embryology (ESHRE)/ Androgen Excess Society (AES) Criteria

→ Anovulation } enough to diagnose PCOS

→ Hyperandrogenism }

Or

Clinically obvious hirsutism — laboratory reports ↑ Testosterone

→ USG – PCOS

: Obesity is not included as criteria

: Can even occur in girls with BMI < 18 kg/m²

Prolapse of Uterus

Classified by

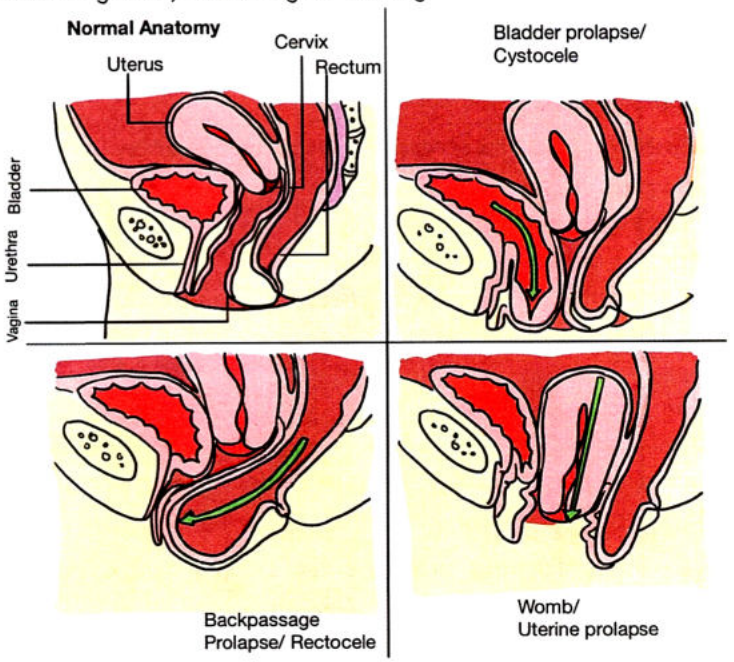
- Shaws system (1-4)
- Jeffcoate system
- **POP – Q** (Pelvic organ Prolapse – Quantification system)-best way to classify

Causes of Prolapse of uterus

- Abnormal conduct of labour [MAIN REASON]
- Connective Tissue disorders (Marfans, Ehler Danlos etc)
- Spina bifida
- ↑ abdominal pressure (because of mass in abdomen, ascites)

Structures involved in prolapse from Anterior to posterior

- Anterior vaginal wall
- Utrethra (Urethrocele)
- Urinary Bladder (Cystocele)
- Uterus
- Rectocele
- Posterior vaginal wall



C/F: usually in older women (55-65y)

Cystocele – difficulty in urinating, urinary retention infection, stone formation

Rectocele – fecolith formation

Others – Decubitus ulcers/ Dependant ulcers on cervix

↓

Chronic Non healing ulcers due to venous congestion by vaginal compression

Mx –Insert **Ring Pessary** after Repositioning.

Disadvantage – temporary management

Advantage – Improves micturition, defecation and decubitus ulcers

2. **Surgical Mx** – Vaginal Hysterectomy

+

Pelvic floor Repair

+ Anterior Colporrhaphy (Tighten ant. Vaginal wall)

+ Posterior Colpoperineorrhaphy

Known as **WARD and MAYO'S OPERATION**

- Anterior colporrhaphy not done properly can cause – stress urinary incontinence (in 2-3 weeks)
- Neglected enterocele – cause Vault prolapse

Stress urinary continence (SUI)

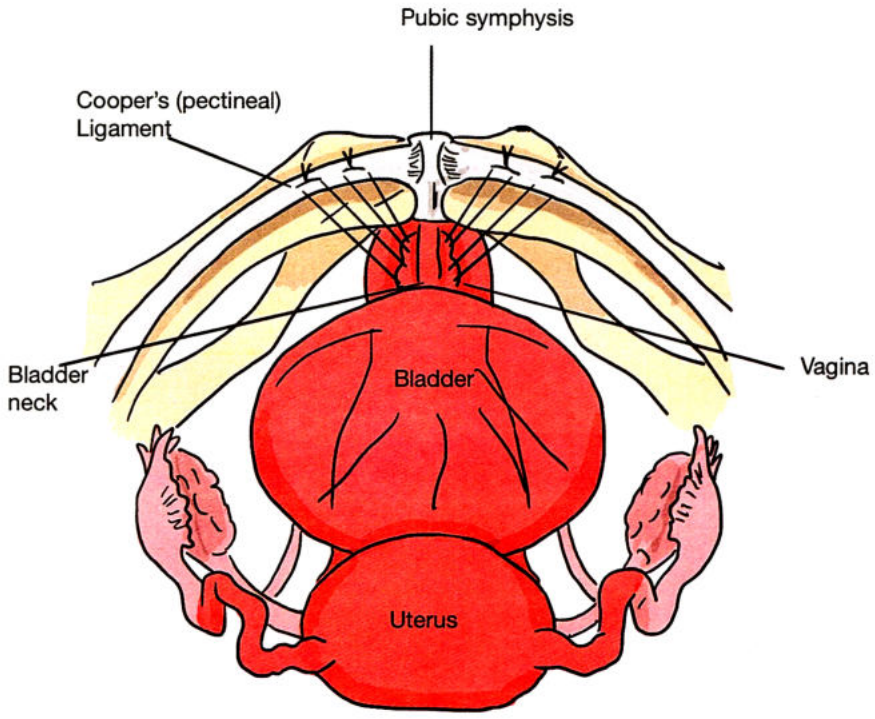
Surgical management

1. **Needle suspension** procedure (pull up urethra)
2. **BURCH Colposuspension**→ up lifting Anterior vaginal wall and attached to coopers ligament
3. **Kelly's STITCH** – bladder neck plication

↓

Paraurethral tissues plicated

4. **TVT** – Tension Free Vaginal Tape



BURCH COLPOSUSPENSION

PID (Pelvic Inflammatory Disease)

- PID stars from cervix (Cervicitis)
  ↓enter
  Uterus (Endometritis)
  ↓
  Fallopian tubes
  ↓
  Pelvic cavity (Endosalpingitis)
- Syndrome presents with- pelvic pain, fever, discharge.
- M/C cause of PID = Chlamydia – (most prevalent; Asymptomatic: Indolent)
- M/C Presenting in OPD = Gonorrhea

Other causes

- Tuberculosis
- Bacterial vaginosis
- Mycoplasma species, ureaplasma

Diagnosis of PID:

Symptoms:

None necessary for diagnosis

Signs:

- Pelvic organ tenderness
  - Pelvic pain
  - Cervical motion tenderness } Triad
  - Adnexal tenderness
- Leukorrhea and /or mucopurulent endocervicitis

Additional criteria to increase specificity of the diagnosis

- Endometrial biopsy showing endometritis
- Elevated C – reactive protein or erythrocyte sedimentation rate
- Temperature higher than 38 C (admission criteria)
- Leukocytosis
- Positive test for gonorrhea or Chlamydia (NAAT) Nucleic acid Amplification Test

Elaborate criteria

- Ultrasound documenting tubo-ovarian abscess
- Laparoscopy visually confirming salpingitis (Best way to confirm PID)

Fitz Hugh Curtis Syndrome

→ Perihepatic adhesions

Treatment of PID: CDC regime

- Inj. Cephalosporin = Gonorrhea
- Doxycycline /Azithromycin = Chlamydia } OPD regime
- Bacterial vaginosis, Anaerobes
  ↓
  Metronidazole/ Clindamycin

→ In Pregnancy: Gonorrhea -Inj. Cephalosporin + Azithromycin

Vaginitis

→ Bacterial vaginosis: Can cause
- Abortion
- PID, Relapses of PID
- Preterm Labor
- Intrauterine death of fetus
- Puerperal sepsis

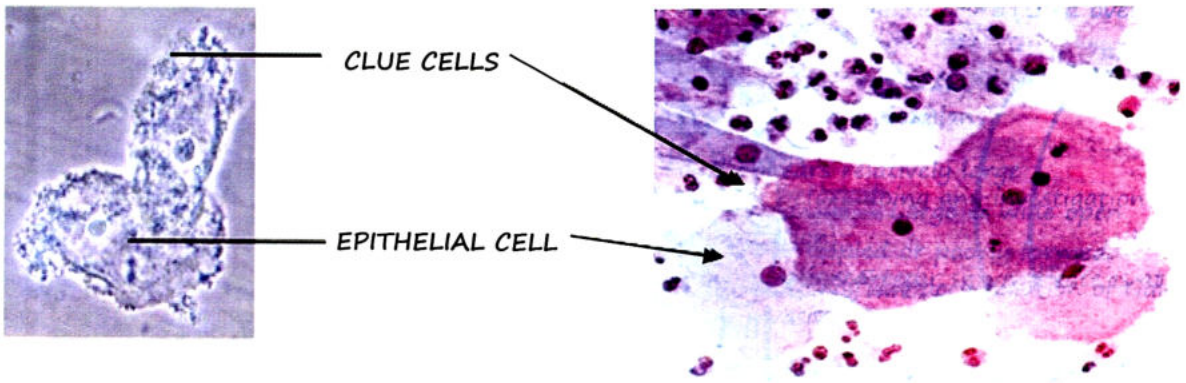
AMSEL'S Criteria>3/4 required for diagnosis
→ Clue cells
→ Creamy discharge
→ Whiff test (Secretions + KOH) → Amines released (Ammoniacal odour)
→ Fishy odour

Diagnosis;-

Microscopy: Vaginal epithelial cells surrounded by bacteria

↓

CLUE CELLS



PCR : Ribosomal RNA
: Metronidazole

Trichomoniasis: Trichomonas vaginalis (flagellate)
: favoured by Alkaline pH
: Syndrome is same as Bacterial Vaginosis
: May show Whiff test positive and Clue cells (as this syndrome has presentation similar to bacterial vaginosis)

Diagnosis
: Microscopy: Flagellate
: Greenish yellow frothy discharge
: Due to severe inflammation -Colpitis Macularis – "Strawberry vagina"
: Severe itching

Rx: Metronidazole
↓
Partner treatment needed (Trophozoite forms of Trichomonas are on the undersurface of prepuce, so even if woman treated, infected male can reinfect her)

Candidiasis

: Occurs in acidic pH.
: Spores responsible for infection
: Mycelia = Responsible for Adherence and invasion of candidiasis into vaginal epithelium.
: Curdy white discharge (Cottage cheese like, classical white discharge per vaginum)
: mostly caused by candida albicans
: In normal women
: Easily treated when uncomplicated

Complicated candidiasis

- Immunocompromised host
    - HIV, pregnancy, Tuberculosis
- Recurrent
- More symptoms
- Difficult to treat
- Caused by "non-albican species"
- Out of proportion itching

Treatment

- Local creams: Butaconazole, Clotrimazole
- Oral: 150mg of Fluconazole

INFERTILITY

→ Inability to have a child after having tried for 1 year
→ Primary infertility (never conceived)
: Secondary Infertility (cannot conceive for 2nd time)

} Duration is 1 year for both

→ Normally, 80% couples conceive within 6 months and 90% couples conceive within 1 year
→ Sub fertility = Conceive >12 months of trying

Fecundibility: Capacity to conceive in 1 cycle
Fecundity: The capacity to have a live birth in 1 cycle

→ If a couple tries and conceives in one cycle, it is fecundibility and if that pregnancy continues for 9 months & gives a live birth it is fecundity.
→ Fecundity is much lower value than fecundibility
→ A person has high fecundity (conceive) but she may have abortion, preterm labor, child loss due to intrauterine death (as in Diabetes). So, pregnancy may not continue for full term.

Causes of Infertility

- Female factors alone = 40-55% cases of infertility
- Male factor alone = 20-30%
- Both male & female factors = 10-40%
- Unexplained infertility = 10-20% cases

→ **Male factors**

- Azoospermia: low sperm count
- Oligoasthenoteratozoospermia (OATZ)

→ **Female factor**

- Oligo/Anovulation – Ovulatory disorders
- Tubal factors

Semen Analysis Parameters (2010 WHO)	Lower Reference Limit /Range
• Semen volume (ml)	1.5 mL
• Sperm concentration	15 million / ml
• Total sperm number	39 (36-42) million/ ejaculate
• Progressive motility	32%
• Total motility	40%
• Vitality (live sperms)	58%
• Sperm morphology	4%
• pH	>/ =7.2
• Leucocyte (10^6/mL)	< 1 million/ ml
• Immunobead test/ mixed Agglutinin reaction (MAR)	<50% (earlier it was 10%)

Sperm morphology = single best prognostic feature of semen analysis

→ **Sperm analysis**: first test to be done in an infertile couple before doing any investigation on the women.

- If man has good semen analysis, then workup of the woman is started

Reason: Easy to collect semen sample; any workup on female is invasive.

Ovulation

→ Basal body temperature ↑ 0.5F

→ Serum Progesterone >3 ng/ ml on Day 21

→ Serial Follicular monitoring (Day 8, 10, 14, 16) on USG: Multiple sittings
[Serial growth and then follicle starts shrinking by day 16]

→ Cervical mucus studies – serially

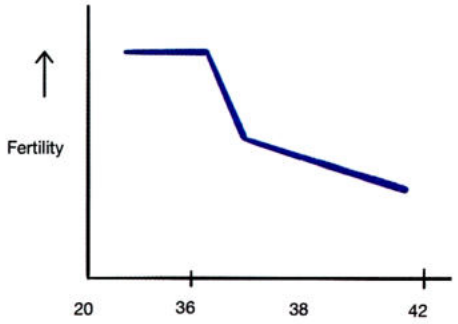
Remember: Thin cervical mucus due to high estrogen and is watery (shows ferning)
: Thick cervical mucus after ovulation under influence of progesterone (loss of Spinnbarkeit and ferning).

:Endometrial biopsy → Secretary changes (best seen on Premenstrual :day 21, 24....)

[How to assess: Day 24: should correspond to → day 24 secretary changes
↓
If day 20 secretary changes seen (lag): Luteal phase defect

Age:

- Best chance of pregnancy in female is 20-25 years of age



In Male: >40 years
Problems
- Increased chances of Disomic sex chromosomes
- Autosomal dominant disorders (like Achondroplasia)

→ Theoretically men can have sperm production till last day of life

Azoospermia

- No sperms in ejaculate

[Aspermia: No semen]

Causes of azoospermia

: Pretesticular Azoospermia

→ Hypogonadotropic hypogonadism
(Pituitary defect, hypothalamic defect, kallmann syndrome)
↓ LH, ↓FSH (required for spermatogenesis)

T/t

- GnRH analogues in pulsatile form
- hMG (menopausal gonadotropins) then HCG for final maturity of sperms

Testicular Azoospermia

→ **Genetic** – Klinefelter's syndrome
- Y – Chromosome deletions: Microdeletions of (long arm of Y chromosome) Yq 11.23

→ 3 types of Azoospermia factors = AZFa,b,c
Most severe is AZFc
- Immotile cilia syndrome (Kartagener syndrome)

→ Congenital

- Cryptorchidism: Undescended testes (higher temperature, ↓ spermatogenesis)

→ Infective (orchitis)

Antispermatogenic agents

- Heat
- Chemotherapy
- Drugs
- Irradiation

Vascular causes

- Torsion
- Varicocele: Severe varicocele (type 3, type 4) = veins are surrounding testes and these have higher temperature especially on left side (left renal, left adrenal veins branches carry toxins)

Treatment

Ovulation induction

- Metformin
- hMG
- FSH

- Clomiphene citrate
- Letrozole

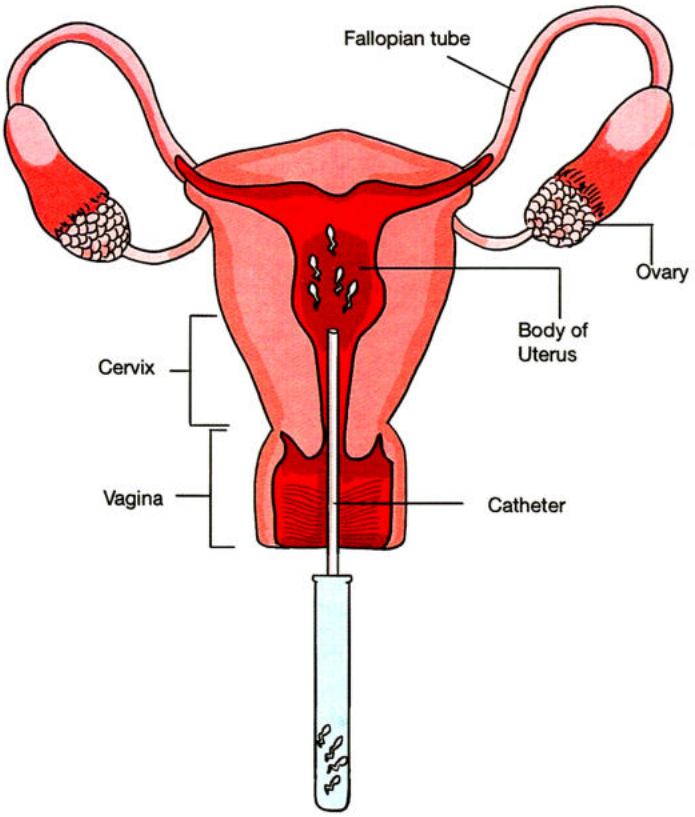
→ ↓Sperm counts, Endometriosis, Cervical factor infertility

-Intrauterine insemination

: Washed sperms are put inside uterine cavity.

: The fallopian tube should be patent, such that oocyte reaches ampulla where fertilization takes place.

: 20-25% chances of pregnancy versus Normal (4-8%)



Assessing Patency of fallopian tubes

- Hysterosalpingography (HSG): Can assess fallopian tubes and uterine cavity
- Laparoscopy + Hysteroscopy

Best assessment of tubal patency

+

(Uterine and tubal) anatomy

+

Biopsy of endometrium can also be done to see adequate secretory function of endometrium

ART: Assisted Reproductive techniques

- External manipulation of both gametes → Sperms, Oocytes

NOTE:- IUI is not ART

In vitro fertilization

→ More eggs are extracted (7-8/ovary)

→ When these follicles get mature, they contain oocytes.

→ Under USG guidance of a transvaginal scope a needle is put directly into follicle and follicles are extracted containing oocyte.

→ Then in laboratory, oocytes are put in test tubes and sperms of husband are used for fertilization = In vitro

In IVF

Step 1: get more eggs (oocytes)

Stimulate ovary by **controlled ovarian hyperstimulation (COH)**→ FSH Injection: Given by long/ short/ flare/ antagonist protocol

→ If uncontrolled stimulation of ovaries → Ovarian hyperstimulation syndrome (OHSS)

20-30 eggs (may reach up to 50-55)

↓

↑↑ estradiol, ↑↑ vascular permeability along with hCG

↓

Fluid shift

↓

Can cause ascites, pleural effusion and pericardial effusion

↓

Patient may even die

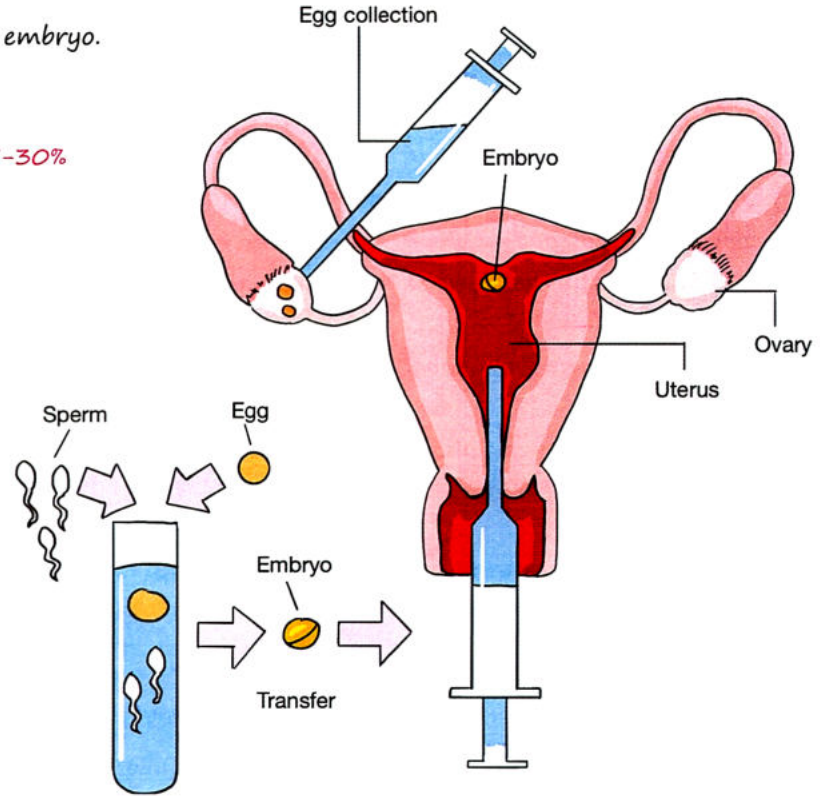
Step 2: Extraction of follicles under USG guidance. Fertilization of oocyte and sperm in test tube (Invitro)

Step 3: Embryos formed in test tube. Put back into uterus for implantation day 3; 8-celled embryo is used. 2-3 are put in uterine cavity.

On day 5 → Blastocyst; we use 1-2 embryo.

Success rate of IVF 40-45%

Take home baby rate after IVF is 25-30%



Dr. Vivek Nalgirkar

Sperm count needed for fertilization in different techniques

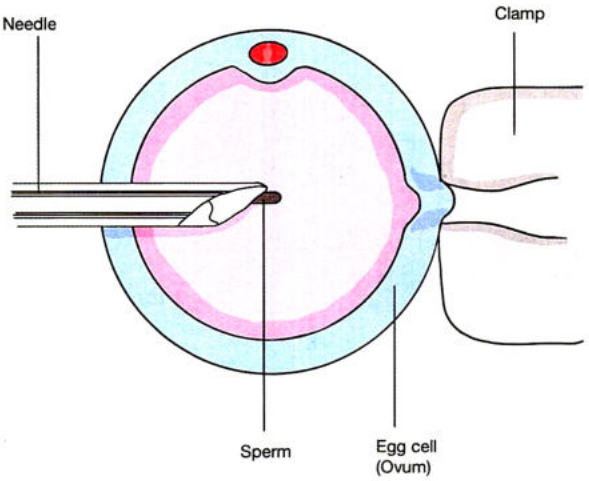
Intra Cytoplasmic Sperm Injection (ICSI)

IUI = 5-10 million sperm count

IVF = 2-5 million sperm count

I.C.S.I. = Very few sperms <1-2 million or Azoospermia:

On aspiration from testes, epididymis – very few sperms



Intracytoplasmic sperm injection (I.C.S.I)

→ Boon for infertile men and women

→ Oocyte is held with a pipette under suction;

In this oocyte a needle is directly injected which is harboring a sperm in it.

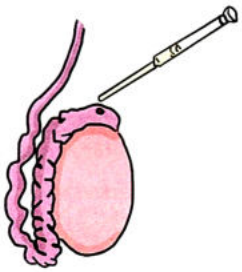
→ Congenital abnormalities, abortion rate: ICSI (4-5%) >IVF (2%)

In case of any IVF, ICSI we are extremely careful in the follow up in 1st, 2nd and 3rd trimester: ultrasound, targeted scanning biomarkers (looking for aneuploidy)

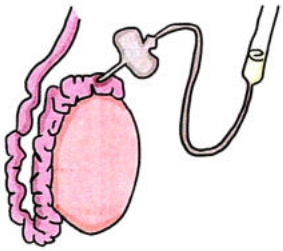
→ Strict obstruction and see if there is any problem in the baby.

Aspiration Techniques:

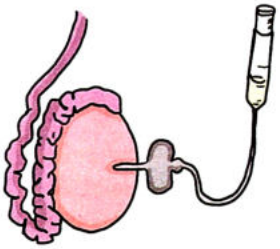
MESA- microsurgical epididymal sperm aspiration (best technique)



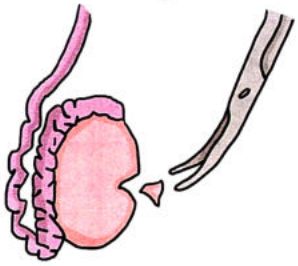
PESA- percutaneous epididymis sperm aspiration



Te.S.A- testicular sperm aspiration



Te.S.E.- testicular sperm extraction



MESA: - An incision made on testes and then sperms are extracted from epididymis under direct visualization with microscopic

Te.S.E:- A chunk of testicular tissue extracted. The tissue with seminiferous tubule is crushed and squeezed; sperms are extracted.

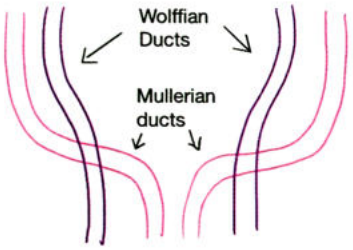
- The sperms best for aspiration for ART are those resting in epididymis
- Epididymis techniques better than testicular techniques.

MESA: - best techniques for aspiration of sperms

Mullerian defects

There are two types of ducts:

1. Mullerian duct (for women): Mullerian genesis occurs in women
2. Wolffian duct (for men)



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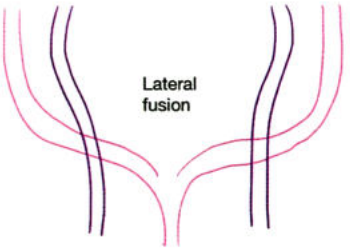

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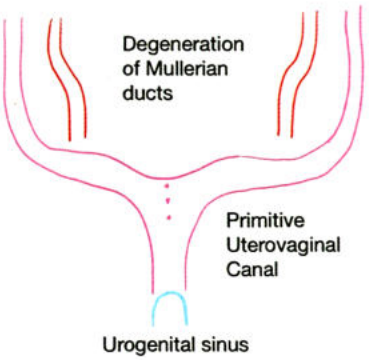

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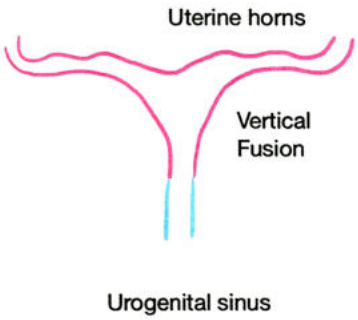


(d)

$4/5^{th}$ of vagina formed by mullerian ducts , $1/5^{th}$ by urogenital sinus.
Mullerian ducts will fuse and form uterus wolffain ducts will get obliterated

Vertical fusion defect

Transverse vaginal septum

→ Uterus, cervix and vagina formed
→ Lower part of vagina also formed
→ But these do not fuse

Vaginal atresia

→ Uterus, cervix formed
→ Vagina not formed
→ Small part of vagina developing from urogenital system formed

Cervico–vaginal atresia

→ Uterus formed
→ Cervix and vagina not formed

Complete Mullerian agenesis

→ Whole uterus, tubes, cervix, vagina not formed
→ Shallow blind vagina present

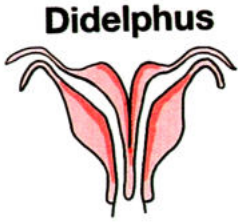
Lateral fusion defects

→ Mullerian ducts of both sides have to fuse in midline, if fusion does not occur or is incomplete, it leads to lateral fusion defects.

Complete Non-fusion of ducts

Didelphys (2 uteri with one/two vagina)

- Bicollis (two vagina)
- Unicollis (one Vagina)



Improper Fusion

Bicornuate

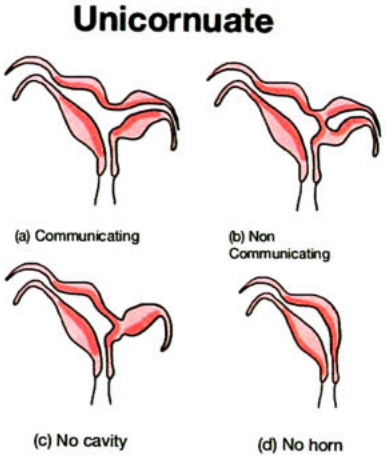
It causes

- Malpresentation
- Pre-term labor
- Abortion

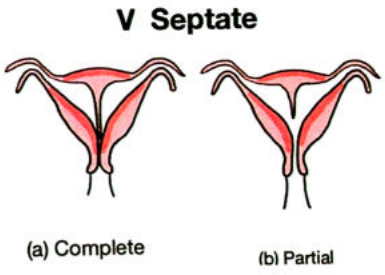
Unicornuate

Unicornuate with a rudimentary horn

- Communicating horn
- Non communicating horn
- In many cases,rudimentary horn which is not Communicating, may have pregnancy in it
- This rudimentary horn pregnancy ruptures
- has presentation similar to an ectopic pregnancy



Septate-**most common Mullerian defect**

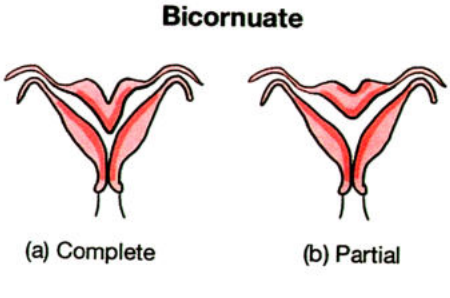


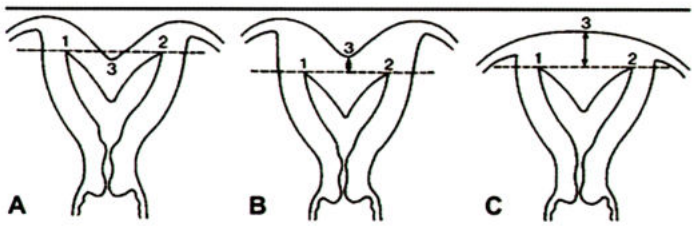
→ How to distinguish bicornuate and septate uterus.

On Hystosalpingography

	Bicornuate	**Septate**
X Rays of bicornuate and septate look similar on HSG		

→ Radiological features to distinguish these





- Distance between 2 horns is more = Bicornuate; When 2 horns are close = septate
- Angle between 2 horns
  obtuse = bicornuate , If acute = septate
- Distance of septum coming below the line drawn between two horns is more in bicornuate and less in septate

→ Best way to distinguish bicornuate and septate uterus –**Laparoscopy + Hysteroscopy**
→ On imaging, **MRI** is best technique

- M/C indication of unification of bicornuate uterus is **Recurrent abortions**
- Bicornuate can have live pregnancy , problem with such pregnancy is: transverse or breech, a live baby is delivered By cesarean.
- Metroplasty done- jones metroplasty, Tompkins metroplasty

Cryptomenorrhea (hidden menstruation): A condition where menstruation is occurring but no bleeding is visualized.
→ Leads to-**Hematometra**
- blood collecting in uterus

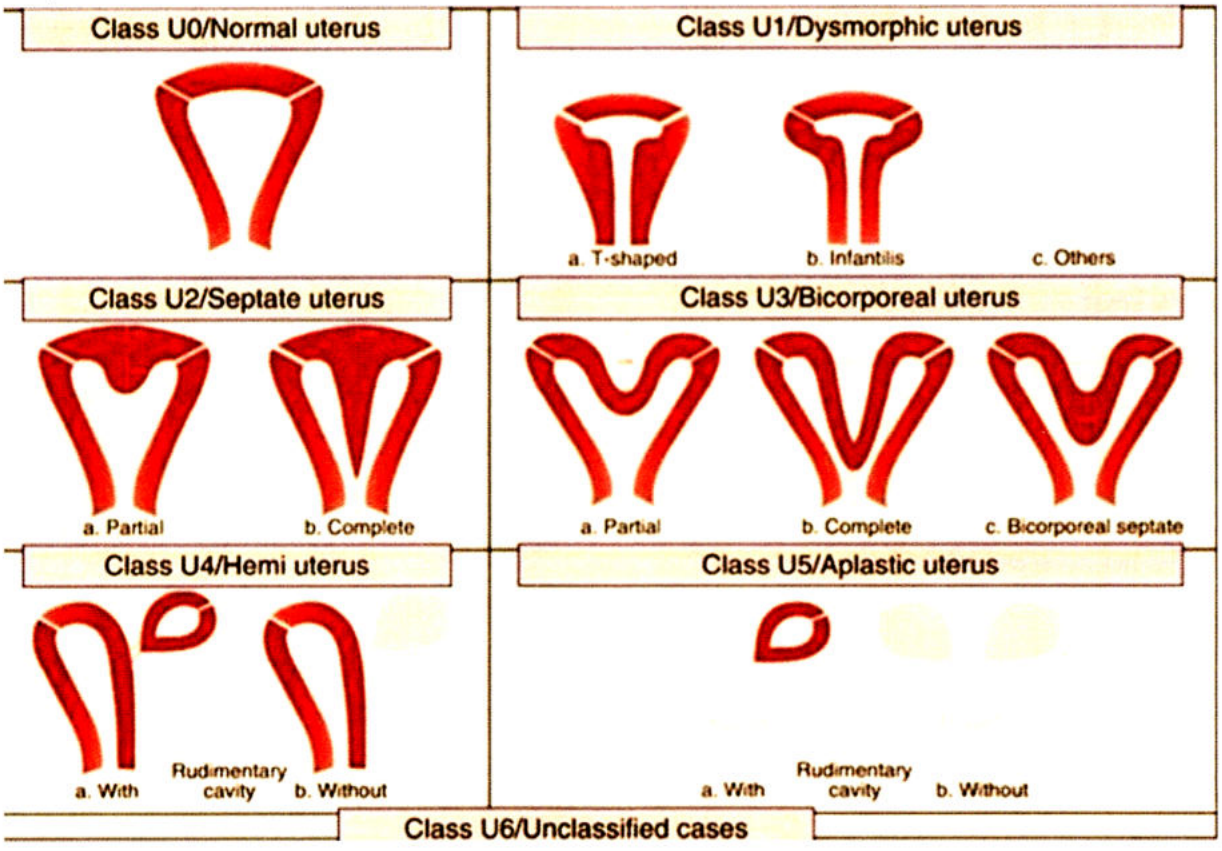
→ Hematocolpos-blood collecting in vagina
Seen in → Vaginal atresia
→ Cervicovaginal atrtesia
→ Imperforate hymen (a cannulation defect of hymen-not a Mullerian defect)

→ Hematometra- bloood collecting in uterus

→ Hemetocolpos- blood collecting in vagina

ESHRE Clasification system for Mullerian defects

(European society for human reproduction in collaboration with congenital uterine anomaly group (CONUTA) in 2013.)



U0- Normal uterus
U1- T shaped or infantile uterus
U2- Septate
U3- Bicorporeal/Bicornuate
U4-unicornuate with/without rudimentary horn
U5- group of anomalieswhere there is complete aplasia of uterus
U6- : Rarer defects which cannot be classified into any system (ESHRE/ American Fertility system, AFS)

E.g.:Mayer Rokitansky- Kuster- Hauser Syndrome (MRKH)

- ESHRE preferred system used for classification
- Vaginal and cervical defects are also included along with uterine defects

Sexual differentiations

Sexual differentiation starts at 3 levels

→ Chromosomal differentiation:female (46 XX); male (46 XY)
↓
Y chromosome has
↓
SRY (sex- determining region Y)
↓
Testes determining factor

→ Gonadal differentiation: Ovary (female); testes (male)
→ Phenotypic differentiation: Vulva (female); Penis (male)

Females internal genitalia formation:-

→ Formation of uterus from Mullerian duct; cervix, fallopian tubes, upper vagina (4/5th)
→ Lower 1/5th of vagina develops from urogenital sinus
→ Ovaries develop from extraembryonic mesoderm and genital ridge

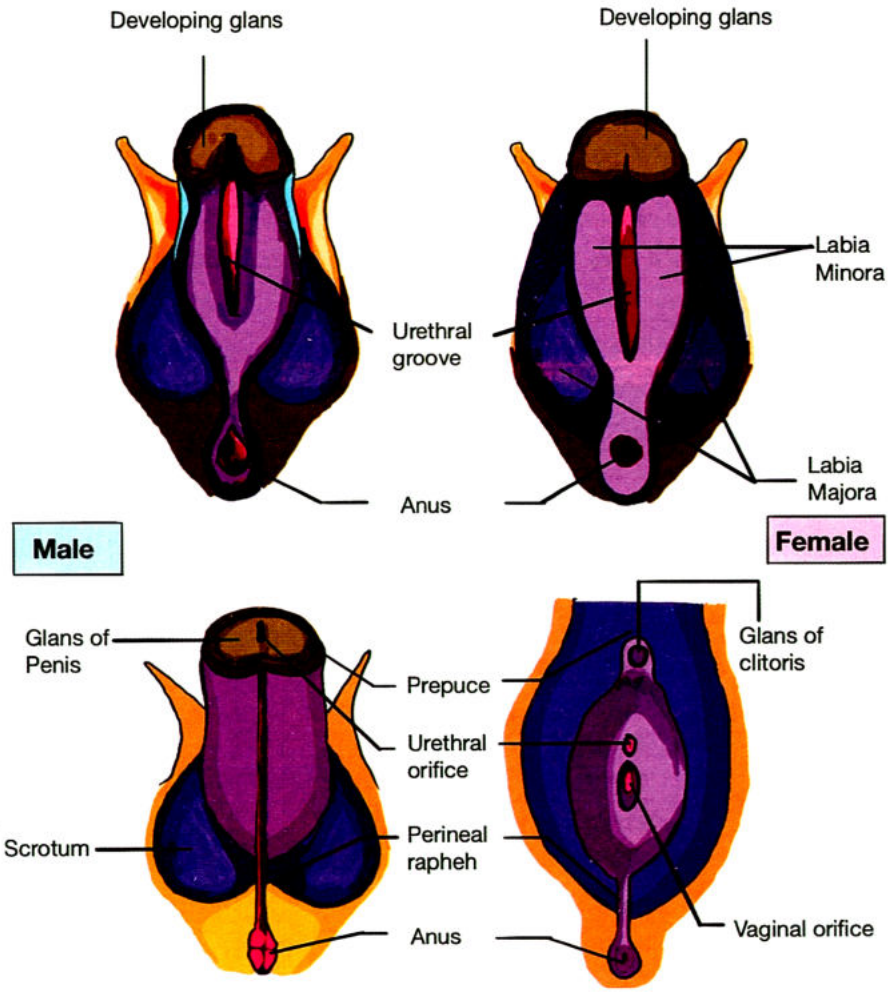
Formation of male internal genitalia

- Epididymis, vas, seminiferous tubules, prostatic part of urethra – develop from Wolffian duct.

Formation of external genitalia

Occurs from

- 1 genital tubercle
- 2 genital swellings
  - Till 6 weeks of intrauterine life these are same in both male and female
- Male fetus is having testes



→Till 6 weeks of intrauterine life 1 genital tubercle and 2 genital swellings are common in male and female

→Then in males testes develop which release androgens

(In females ovaries develop, absence of androgens –female differentiation)

→In males androgens produced:

- These fuse the genital swellings to make scrotum
- increase length of genital tubercle to form penis
- cause descent of testes into scrotum sac (pull down is due to gubernaculum testes)
- **Androgens make a male**
- Urogenital sinus will fuse into base of scrotum

→ Females

- **Absence of androgens in intrauterine life make woman**
- Genital tubercle will remain small and form clitoris
- Genital swellings will remain separate to make labia majora and labia minora
- Urogenital sinus comes and fuses between labia to form lower 1/5th of vagina.

Imp Point: -Default human sexuality is female form under influence of androgens male is formed.

Testicular feminization syndrome (TFS)/Androgen Insensitivity syndrome (AIS) 46XY

Testes → Androgen

↓

Not working

↓

Absence of androgen

- **Boy is born like a female**
- Excess androgen which is not converted to active form produces dihydrotestosterone

↓

When dihydrotestosterone formed, not utilized by end organs

↓

These androgens and dihydrotestosterone converted into estrogen

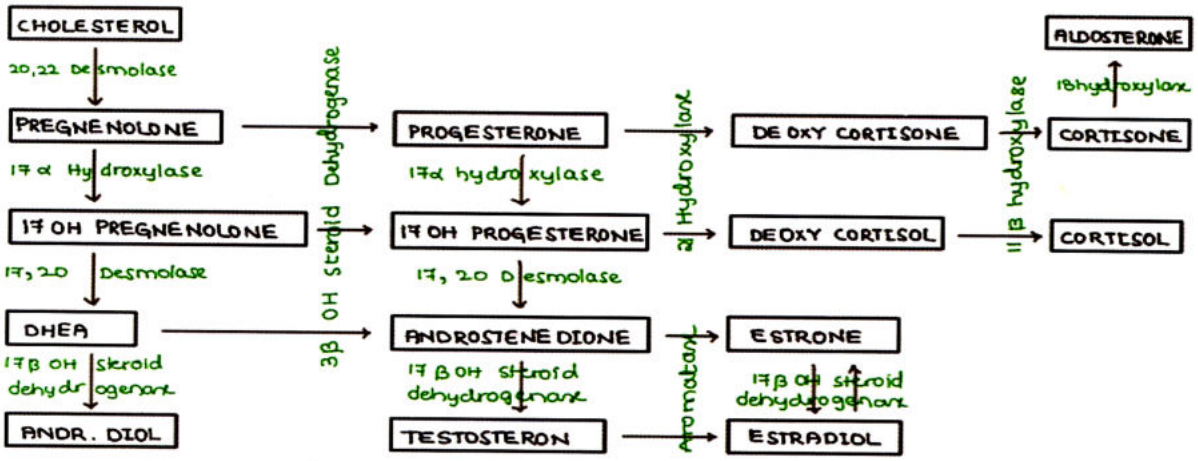
↓

Cause glandular development of **breast**

→ TFS- person looks almost like a normal female

Congenital adrenal hyperplasia (CAH):46XX

- Most common enzyme defect in CAH is **21 Hydroxylase deficiency**
- Cholesterol breaks down in adrenal glands to form androgens and steroids (aldosterone, corticosterone and cortisol)



In deficiency of 21 Hydroxylase in intrauterine female

↓

No feedback to brain due to absence of steroids

↓

Hypothalamus stimulates pituitary

↓

More ACTH release from pituitary

↓

Stimulates adrenal glands to recruit more cholesterol

↓

Extra androgens in a female

↓

Genital swellings fuse and form scrotum; growth of genital tubercle forms penis

↓

Hirsutism (In late onset)

Gonadal dysgenesis in males

MIF (Not formed) ← Testes (Not working) → Androgen (not formed)

→ MIF (Mullerian Inhibiting Factor) not formed, Mullerian exhibition occur forming uterus, cervix, vagina with lower 1/5 vagina from urogenital sinus.

→ known as **Swyer syndrome (46 X Y; female)**

Gonadal dysgenesis in female

- Ovaries not working properly, No estrogen
- No androgen production
- Female is formed

→ After birth, no estrogen

↓

No proper development of uterus

(Hypoplastic uterus)

↓

Primary amenorrhea

→ Female who is lesser feminine

E.g.:Turner syndrome (45, XO)

Note:-

→In gonadal dysgenesis in male and female both = external sexuality is female

→ 46 XX = female; 1 Barr body is present;46XO = lesser feminine female, No barr body.

Turner syndrome(45 X O):

C/F

- Hypoplastic uterus
- Primary amenorrhea
- Short stature
- A shield chest
- Low set ears
- Low set hairline
- Coarctation of aorta
- Lymphedema
- Cubitus valgus
- Lesser feminity
- Normal intelligence

Klinefelter's syndrome(47 XXY):

C/F

- Tall
- Gynaecomastia
- Obesity
- Testicular atrophy
- Infertility
- Mental retardation

Abnormal uterine bleeding (AUB)

→ Any bleeding away from normal period

Normal menstruation:

- 2-7 days bleeding
- Once in 21-35 days
- Bleeding upto 35 ml

Bleeding >80 ml = **menorrhagia**

Bleeding <10 ml = **Oligomenorrhea**

Management of AUB

First line drugs for AUB(both)

- **NSAIDS**: These inhibit vasodilators (prostaglandins)→ vasoconstriction→bleeding reduces
  :Reduce pain and bleeding also (advantage)
- **Tranexemic acid**- It inhibits fibrinolysis prevents re-bleeding

Hormones

Progesterone

-In form of Medroxyprogesterone acetate (MPA)

Dose: 5-10 mg tab 2-3 times in a day for 10-15 days which stops bleeding in 2-3 days, further progesterone stabilizes endometrium can be given upto 21 days, when she stops progesterone, she gets period in 2-8 days (monthly)-Regular periods

Estrogens: given if progesterone does not work

- Estrogen makes new glands ; replace degenerating glands

Which are bleeding

↓

These new gland shed and bleed

↓

Causing more bleeding

→ Dosage: Tablet

- i.v. Estrogen 25-40 mg

Combined OCPs (COCPs) - estrogen (low dose 0.03 mg)& progesterone

No LH, FSH formed – no ovulation

- "Artificial menstruation"

Dosage – tablets

2-2 tablets x 3 days
1-1 tablet x 5 days
1 tablet x 13 days

} based on patient to patient

When stopped after 21 days there is bleeeding

Management of menorrhagia

- Resuscitate
- IV fluids
- Blood (if having anemia)
- Iron tablets started
- For management of bleeding COCPs started

Danazol /Testosterone: tablets /injections

- Cause atrophy of endometrium (also used in endometriosis)

DMPA can also be used (150 mg/ 3 month) inj.
(Depot Medroxy progesterone acetate)

- Atrophy of endometrium
- Progesterone stabilizes endometrium

GnRH analogues – downregulation of pituitary

Levonorgesterol IUCD: Local progesterone causes atrophy of endometrium & stabilizes it.

→ Good for long term

Surgical methods to control bleeding

- "Dilatation and curettage"
- Therapeutic curettage

Uterine artery embolization

→Uterine artery can be embolized by polyvinyl alcohol particle (PVA)

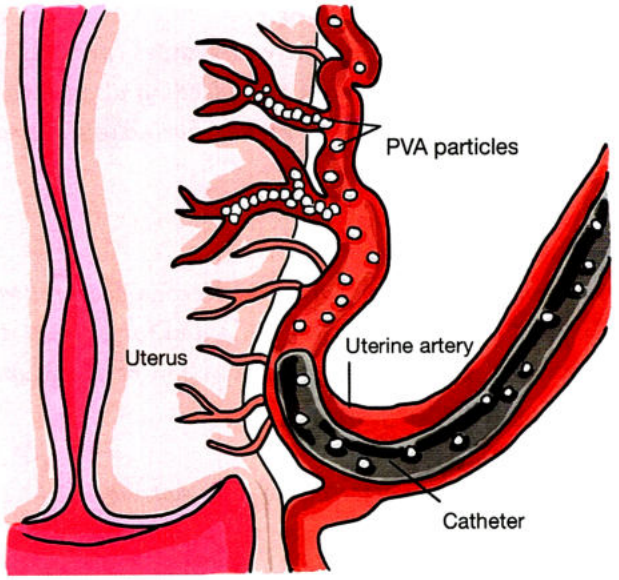
→ Stops bleeding

Tamponade of uterine cavity: - Uterine compression by tamponade

Endometrial ablation:-

Scarring of uterine lining, never bleed again; done in females >35 years of age.

- Amenorrhea (due to fibrosed endometrium although ovaries are functioning)
- Not menopause
- OPD procedure; can avoid need for hysterectomy which is a major surgery.



Hysterectomy: last resort

- Major surgery requiring admission
- Blood transfusion needed in many cases

Complications of hysterectomy can be

- Wound dehiscence, infection
- Deep venous thrombosis
- Can even lead to death (worst case scenario)

Cause of AUB

- Tumors – Ca. cervix, Ca. endometrium, fibroid
- Infections – PID
- Pregnancy related- abortion, secondary PPH, Ectopic pregnancy
- Systemic causes:- hypothyroidism, hyperprolactinemia
- Coagulation defects e.g.- ITP, von-Villebrand disease
- Drugse.g.- Heparin, irregular OCP intake, IUCD

PALM COEIN

Classsification for causes of AUB

- Polyp
- Adenomyosis
- Leiomyoma
- Malignancy and hyperplasia
- Coagulopathy
- Ovulatory dysfunction
- Endometrial causes
- Iatrogenic
- Not yet classified

Fibroids

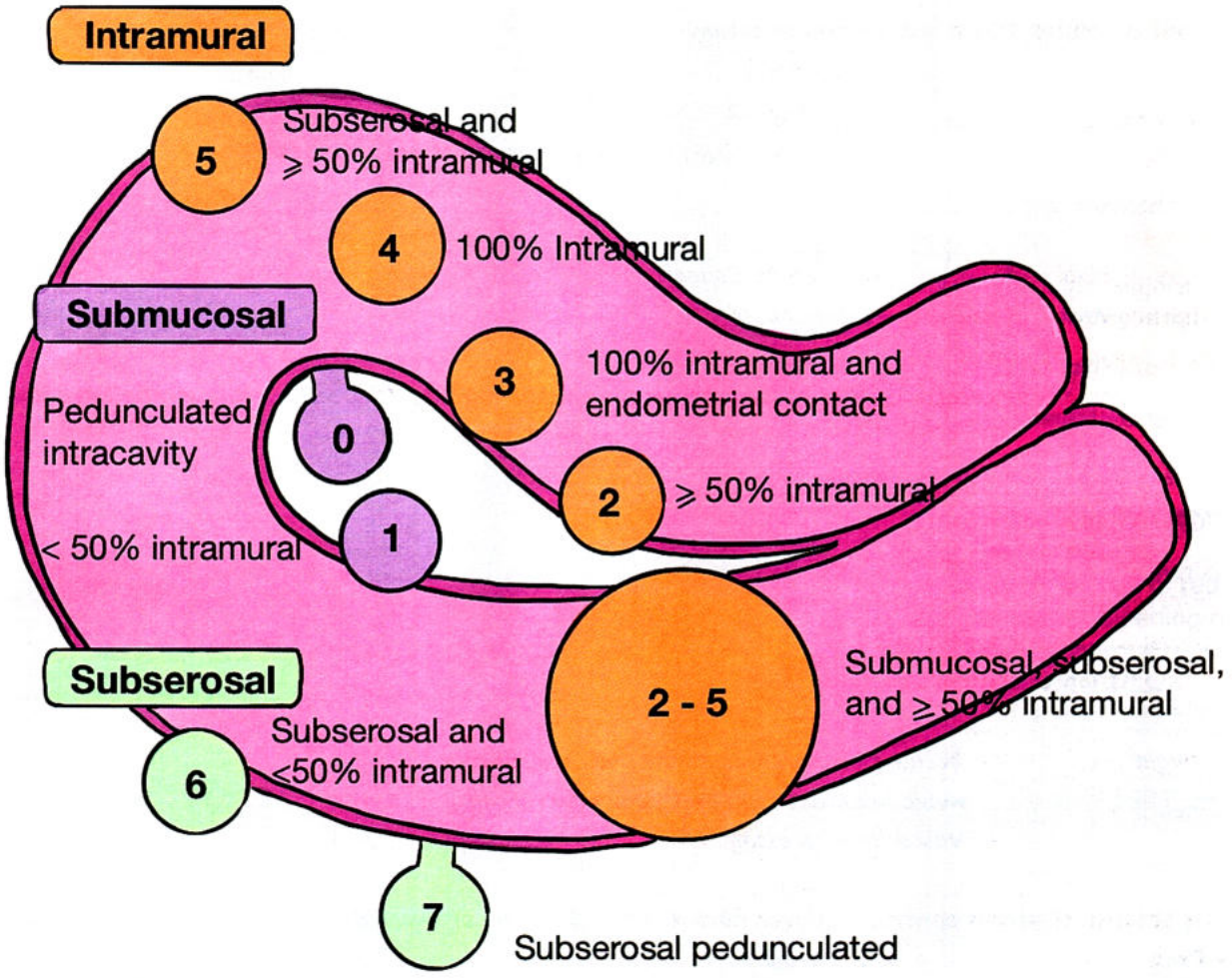
- Monoclonal smooth muscle tumors
- Make whorls of smooth muscle cells
- Always starts intramural

With contractions of uterus fibroids can be pushed to

- Surface → Subserous
- pushed inwards → Submucous

FIGO classification of fibroids

(Federation of international gynecologists & obstetricians)



SM – Submucosal	0	Pedunculated Intracavitary
	1	<50% intramural
	2	≥ 50% intramural
O – Other	3	Contacts Endometrium; 100% intramural
	4	Intramural
	5	Subserosal ≥ 50% intramural
	6	Subserosal < 50% intramural
	7	Subserosal pedunculated
	8	Other (specify e.g. cervical, parasitic)

Hybrid leiomyomas (impact both Endometrium and serosa)	Two numbers are listed separated by a hyphen. By convention, the first refers to the relationship with the Endometrium while the second refers to the relationship to the serosa. One example is below	
	2-5	Submucosal and subserosal, each with less than half the diameter in the endometrial and peritoneal cavities, respectively.

Etiology

- ↑ Estrogen, ↑ progesterone
- Genetic factors 40% = 12;14 translocation
  Deletion 7
  Trisomy 12
- Increased growth factors: TGF-Beta, VEGF, basic FGF (fibroblast growth factor)

Epidemiology

- More in black women as compared to Caucasians
- More common in red meat eaters;obese
- M/C tumor of uterus
- M/C cause of hysterectomies (70% of hysterectomies have fibroids 30- 35% of all women have fibroids).

Symptoms

- Menorrhagia
- Pain
- Infertility
- Bowel & bladder symptoms
- Compression & compaction symptoms

Menorrhagia – because of increased surface area of fibroid
- more endometrium and poor contractility of uterus
-↑ Vasodilator prostaglandins

Pain – is because of uterus contracting over fibroids to push it out or inwards
- Pressure symptoms of a large fibroid

Infertility - because of tubal occlusion or uterus contracting to push fibroid out – dislodges embryo or even prevent implantation

Medical management

- ↓Bleeding: drugs used
  - NSAIDs, Tranexemic acid
  - Hormones – Inj. DMPA;COCPs
- ↓Size
  - GnRH analogues
  - GnRH antagonist
- LNG-IUCD: ↓bleeding
- Uterine artery embolization

Surgical management

- Myomectomy
  - Laparoscopic
    - Serosa
    - Intramural
  - Hysteroscopic – sub mucosal fibroids
- Hysterectomy
  - Laparoscopic hysterectomy
  - Laparotomy

Laparoscopic myomectomy

- Size 5 cm or less ; 3-4 fibroids
- Or 1 fibroids <15 cm

To reduce bleeding during myomectomy
medical management is used prior to surgery
Intraoperatively

- **Drugs**
  - Vasopressin(Pitressin)
    ↓ (intra fibroid)
    Reduced vascularity to fibroid; lesser blood loss
  - Methylergometrine (causes uterine contraction)
  - Prostaglandins (Intramyometrial to decrease blood loss

-**Uterine artery clamping**

- Tourniquet over uterine artery to reduce blood supply
- Hypotensive anaesthesia

Prerequisites for myomectomy:-

- Our main aim is to save uterus to preserve fertility
- Semen analysis should be good
- Hb >10 gm.%
- Minimal incision on uterus
- Minimum or no handling of fallopian tubes

Note:-

→ Small fibroids, asymptomatic ≤5cm: No treatment needed
→ Small, symptomatic (pain, bleeding, infertility) - treatment needed
→ Large with symptoms: - treatment necessary
→ Large even asymptomatic: - treatment necessary

Large fibroids: - Can undergo degeneration, can cause compression and compaction, cause bladder & bowel symptoms
So, treatment is necessary whether symptomatic or asymptomatic

Degeneration of fibroids

- M/C- Hyaline
- M/C in pregnancy: Red degeneration (due to infarction) - In 2nd trimester most of the time.

T/T conservative – pain killers and rest

- Lipoid degeneration , calcific degeneration, sarcomatous (<0.5% rarest)degeneration
- Null parity- factor which predisposes to fibroid uterus
- Classically, females with 2-3 children have lesser chances of fibroids
- **Mifepristone**: - Progesterone antagonist ; Rate of fibroid growth decreases d/t lesser progesterone stimulation

Endometriosis

- Ectopic endometrium
- Due to retrograde menses – **Sampson's theory**
- M/C site – ovary, pouch of Douglas
- 70-90% females have retrograde menstruation

↓

Most have very good immunity

↓

Deposits get cleared by macrophages

[Poor immunity, ↑ estrogen]

↓

10% develop endometriosis

Diagnosis

- USG
- CA 125
- Best: Direct visualization by laparoscopy

↓

Biopsy of deposits

↓

Endometrium on HPE

↓

Hemosiderin laden macrophages

- Endometriosis is seen as – blue spot or powder burn lesion
- Adhesions : because of healing

Symptoms

- Pain abdomen
- Dysmenorrhea (congestive type)
- Dyspareunia
- Infertility (due to adhesions)

Infertility is due to

- Altered tubo-ovarian relation
- Reduction of oocyte quality
- Adhesions

-Embryotoxic action of deposits on embryo

- Menorrhagia (due to ↑ estrogen)

Treatment of endometriosis

Surgical

- Removal of deposits
- Chocolate cystectomy (deposits in ovary)
- Adhesiolysis of adhesions
- Ablate the deposits by laser or thermal methods

Medical management

Key of management in endometriosis because 60-70% cases recur. (over 6 months to 1 year)

- Combined OCPs – limit endometriosis
- Danazol: atrophy of endometrium
- Inj. DMPA 150 mg /3 months- atrophy of endometrium
- GnRH analogue- depot or continuous form

E.g.: Leuprorelin, Nafarelin, Buserelin

- Normally GnRH from hypothalamus is secreted in a pulsatile manner (Once per 90 minutes)
  ↓
  This stimulates pituitary
  ↓
  Release LH, FSH
- GnRH analogues continuous or depot form inhibits pituitary (non-responsive) – Down regulation

- No LH, FSH – No estrogen, no progesterone
  ↓
  Atrophy of endometrium

Basic management of endometriosis

Stop the periods

Endometriosis
↓
Surgical removal
↓
Medical suppression

Advised to complete Family
As soon as possible

Family complete
↓
maintain on low dose COCPs

Combined OCPs

4 Packets → 84 days → Break → Period → again 4 packs for next 84 days

Mifepristone (RU- 486):

- Has anti-endometrial cell proliferation action
- Anti – glucocorticoid action (steroids known to proliferate the deposits)

TYPES OF HYSTERECTOMY

1. EORTC – GCG	
Type I	**Simple hysterectomy**
Type II	**Modified radical hysterectomy** → Ureters are dissected up to the point they enter the bladder; → Uterine arteries are sectioned and ligated at the medial half of parameters; → Proximal uterosacral ligament resection; → The medial half of the cardinal ligaments is excised; → 1-2 cm from the upper portion of the vagina is removed.
Type III	**Radical hysterectomy** → Removal as far as possible from the uterosacral ligaments; → Parameter is resected as near as possible to the pelvic wall; → Uterine vessels are ligated at the origin; → 1/3 of the upper vagina is removed
Type IV	**Extended radical hysterectomy** → Similar to type III but with the removal of ¾ of the vagina and paravaginal tissue counterpart.
Type V	**Partial Pelvectomy** → Terminal ureter or a portion of the bladder or rectum is resected together with the uterus and parameters (Supralevatorial)

2. Piver – Rutledge smith	
Class I	**Extrafascial hysterectomy** → The identification of ureters through transparency and avoiding the ureter injury by running them outside the operator field, without a dissection; → the uterine artery is laterouterine sectioned and ligated; → Uterosacral and cardinal ligaments are not removed. → No vaginal portion is excised;
Class II	**Modified radial hysterectomy (Wertheim)** → Ureters are dissected in the paracervical region but are not resected from the pubovesical ligament → The uterine arteries are sectioned beside and medial to the ureter; → Uterosacral ligaments are excised midway from their sacral insertion; → Resection of the cardinal ligaments up to their medial half; → Removal of the upper third of the vagina; → Pelvic lymphadenectomy
Class III	**Classical radical hysterectomy (Meigs)** → Complete dissection of ureters pubovesical ligaments except for a small part where the umbilical bladder artery is situated to the level of their penetration into the bladder; → Uterine arteries are cut off at the origin of a hypogastric region (a, umbilical bladder); → Uterosacral ligaments are excised at their sacred insertion; → Cardinal ligaments are resected as close to the pelvic wall; → Removing the upper half of the vagina; → Routine pelvic lymphadenectomy 3
Class IV	If differs from the previous class according to the following aspects which give a higher radicality; → Complete dissection of the ureter from the pubovesical ligament → Umbilical vesical artery is sacrificed. → Removal of the ¾ of the upper vagina;
Class V	It is more radical than the previous class with the addition of → Excision of a portion of the ureter or bladder which is invaded and then the reimplantation of the ureter into the bladder.

3. Querlieu & Morrow	
Type A	**Extrafascial hysterectomy** → Identification and palpation of the ureters without the dissection of the ureteral layer → the uterine arteries, the uterosacral ligaments and cardinal ligaments are resected as close as possible to the uterus; → The removal of a vaginal portion as small as possible (<10 mm);
Type B1	→ The ureters are deperitonized and rolled to the lateral side; → Partial resection of the utersocral and vesicouterine ligaments → Section of paracervical tissue at ureteral tunnel level. → At least 10 mm of the vagina are measured from the cervix or the tumor → Without removal of lateral paracervical lymph nodes.
Type B2	→ The ureters are deperitonized and rolled to the lateral side; → Partial resection of the uterosacral and vesicouterine ligaments → Section of paracervical tissue at ureteral tunnel level. → At least 10 mm of vagina measured from the cervix or the tumor; → Removal of lateral paracervical lymph nodes;
Type C1	→ Ureters are fully mobilized; → Sectioning of utero – sacral ligaments at the level of the rectum; → Sectioning of vesico – uterine ligaments at the level of the bladder; → Complete resection of paracervical tissue; → 15 to 20 mm from the vagina resected towards the cervix or tumor and correspondent paracolpos. → With the preservation of the autonomic nerves.
Type C2	→ Ureters are fully mobilized; → The sectioning of utero – sacral ligament at the level of the rectum; → The sectioning of vesico – uterine ligament at the level of the bladder; → Complete resection of paracervical tissue; → 15 to 20 mm from the vagina resected towards the cervix or tumor and correspondent paracolpos; → Without the preservation of the autonomic nerves;
Type D1	→ Full resection of the paracervical tissue up to the wall of the pelvic bone together with the hypo gastric vessels, exposing the sciatic nerve roots; → Ureters fully ambulant
Type D2	→ Full resection of the paracervical tissue up to the wall of the pelvic bone together with the hypogastric vessels;exposing the sciatic nerve roots; → Ureter fully ambulant → Resection of muscles and adjacent fascia.

PAEDIATRICS